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VOL. XLII. DECEMBER, 1933. No. 4.

LXXVIII.

THE VENOUS CIRCULATION OF THE PETROUS BONE AND ITS CLINICAL SIGNIFICANCE.

SIMON L. RUSKIN, M. D.,

NEW YORK.

A NEW SYNDROME OF PETROSAL INVOLVEMENT.

Most of the attention devoted to the temporal bone has hitherto been focused upon the mastoid process, its diseases, and the methods for its control. This has fortunately progressed to a point where surgical technic is relatively standardized and efficient. This development, however, required a preliminary adequate demonstration of the regional anatomy. Recent researches are bringing the petrous bone in line for the same surgical development that the earlier studies did for the mastoid. This could not be better expressed than in the words of Sir Charles Ballance at a time when mastoid surgery was at the same threshold. "The safety that comes of boldness and skill in surgical work cannot be attained without accurate anatomic knowledge. The surgical treatment of most of the bones of the skeleton had long been scientifically and successfully carried out before the minds of surgeons were awakened to a clear perception of the fact that in disease of the temporal bone the same systematic surgical treatment was equally applicable and even more urgently called for. The earlier operations on the temporal bone lacked any real basis

of anatomic and pathologic knowledge and in consequence were halting in execution and failures in result." It is the energetic pursuit of the anatomic factors of the petrous bone that will develop the early diagnosis of involvement and the adequate surgical technic.

It is the object of this work to show first that the earliest sign of involvement of the petrous bone is disturbance of circulation in the venous network of the petrous bone; second, that there are two outstanding syndromes, one described by Gradenigo, with involvement of the abducent nerve, and orbital pain; the other represents the syndrome which I am here suggesting, characterized by edema of the lower lid on the same side, temporomaxillary orbital pains and trismus.

The above mentioned venous syndromes present the early circulatory indications and usually precede the suppurative state. It is still a matter open to further study, whether suppuration of the petrous bone always follows from direct extension of cellular groups, or is the result of circulatory disturbance leading to bone coalescence at a point remote from the original infection.

The petrous bone may literally be said to lie in a venous bed. The posterior surface is completely encircled, laterally by the sigmoid sinus and bulb, superiorly and inferiorly by the petrosal sinuses and medially by the cavernous sinus. Inferiorly, the jugular bulb has extensive contact with the petrous bone. Anteriorly is the pterygoid plexus and the closely associated ophthalmic veins. The great significance of this relationship lies not only in the question of involvement of the petrous bone, but also in the fact that this venous bed likewise supplies the important structures surrounding it, and thus ties involvement of the petrous bone to intracranial complications. These points are demonstrated clearly by the accompanying dissections. In order to understand the larger venous sinuses it is necessary to consider the entire venous mechanism and its most detailed construction. This has, in part, been admirably described by Macewan (page 23). According to Macewan, the inferior petrosal receives blood from the cavernous sinus, the inferior cerebellar veins and those from the aqueduct of the cochlea, the vestibule and the fossa subarcuata. It pours its blood into the jugular bulb occupying the anterior compart-

ment of the jugular fossa. It receives small veins through the foramen lacerum medium. Pietrantoni, however, includes certain of the tympanic veins.

The superior petrosal extends from the cavernous sinus to the junction of the lateral with the sigmoid sinus. It receives blood from the inferior cerebral and superior cerebellar veins and small branches from the tympanum, many of which issue through the petrosquamosal suture, the remainder passing through the bone directly into the sinus.

The basilar plexus over the basilar process of the occipital opens into the inferior petrosal on both sides and into the anterior spinal veins below. According to Macewan, most of the mastoid veins pass into the sigmoid sinus. The veins of the petrous bone pass partly into the sigmoid and partly into the petrosals.

The tympanic veins we shall trace in greater detail subsequently.

The pterygoid plexus is situated around the external pterygoid muscle, chiefly between its two heads, which converge to be inserted into the pterygoid fossa beneath the condyle of the lower jaw. The intracranial blood is poured into the pterygoid plexus by the middle meningeal veins through the foramen spinosum, by the Vesalian piercing the sphenoid and communicating directly with the cavernous sinus and by the communicating ophthalmic vein which passes through the sphenomaxillary fissure. Besides receiving many muscular branches, it has an intimate relation with the venous circulation of the nose through veins from the nasal fossa pouring their blood into it through the sphenomaxillary vein. The blood from the pterygoid plexus is conveyed through the facial vein and the internal maxillary veins into the jugulars, external and internal.

Bearing these larger venous structures in mind, let us now turn to a consideration of the finer venous structure of the petrous bone itself, as well as the venous supply in relation with the various nerves of the petrous region and their pathologic and clinical aspects.

The anatomic and pathologic observations on these points are exceedingly few, because of the difficulties in the preparation of anatomic tissue for proper study. The pathologic demonstrations

are still more difficult because the preparations that become available are those that have suffered bone coalescence and suppurative destruction. Since this paper deals primarily with a condition that either resolves spontaneously or passes over into a different pathologic state—that is, suppuration, the conclusions must be arrived at by matching careful clinical observations with available anatomic knowledge. It is by this method that it has been possible to draw the conclusions here presented.

In considering the paths that are followed by inflammatory processes spreading from the tympanic cavity to the cavernous sinus, five routes have been suggested. First, by the small vessels which unite directly the venous circulation of the bony wall of the tympanic cavity with the pericarotid plexus that extends to the cavernous sinus. The thrombophlebitis of these little veins would permit the spreading of the morbid process from one cavity to another. In the more serious cases there exists a true osteitis of the osseous wall of the carotid canal. It is necessary, however, to bear in mind that in some cases the lesion of the pericarotid plexus are secondary to those of the cavernous sinus.

A second route exists in the direct communication between the venous circulation of the tympanic cavity and the inferior petrosal sinus across a group of veins clearly distinguished from the internal auditory veins and from there to the cavernous sinus.

A third pathway lies in the communication of the small veins of the tympanic cavity directly with the superior petrosal sinus. According to Davis (1911), Rodger (1922), Hemmeon (1922) and Shemelay (1923), this manner of propagation is relatively frequent.

The fourth route follows the structure of the various cell groups which tend to extend from the tympanic cavity to the apex of the petrous bone, both peritubal and perilabyrinthian. The mucosa of these cells conduct the veins from cell to cell towards the tip. My preparations show this pathway very distinctly. Pietrantoni has been able to follow these veins from one cell to another. From the apex of the petrous bone they can then spread to the cavernous sinus. His anatomic demonstration tends to bear out these hypotheses, which he has advanced.

Pietrantoni made a study of a series of sections of an injected temporal bone and found that the inferior petrous sinus is connected with the venous circulation of the tympanic cavity. Numerous groups of veins pass out from the submucous plexus of the lower and median walls of the tympanic cavity not only indirectly, as is commonly believed, but directly by means of many veins. Some of them traverse the bone plate of the lower wall and empty, part of them directly into the sinus of the jugular, and part of them, after running a little way toward the midline, anastomose with the peripharyngeal plexus, while others remain in the posterior surface of the petrous bone and run toward the midline and upward. Of this group, the most anterior ones anastomose with the internal auditory vein, while the posterior ones, which Pietrantoni considers leave the bone to the median side of the jugular sinus, empty directly into the inferior petrosal sinus at the level of the internal auditory foramen. His sections show that independently of the internal auditory vein, there are venous trunks which pass from the tympanic cavity directly into the inferior petrous sinus. The above described veins run in the posterior surface of the pyramid of the temporal bone, while those that establish a venous connection between the tympanic cavity, the tip of the petrous bone, the sixth pair of nerves and the Gasserian ganglion are near the anterior surface of the bone. The system of labyrinthian cavities lies between the two posterior thirds of the tympanic cavity and the tip of the petrous bone. Between the anterior third of the tympanic cavity and the tip of the petrous bone there is a tract of compact bone tissue traversed by the carotid canal. This part of the temporal bone shows a more or less compact structure, depending on the individual, and sometimes in the adult it shows a true pneumatization like that of the mastoid process. This tract of bone tissue is traversed by a fine reticulate plexus of veins which externally is continuous with the submucous venous plexus of the tympanic cavity and internally joins the many venous trunks of the tip of the petrous bone. The posterior ones of these empty into the inferior petrous sinus a little before it unites with the cavernous sinus; the median ones anastomose with the pericarotid venous plexus and the anterior ones having united with the vascular plexus which sur-

rounds the Gasserian ganglion empty into the cavernous sinus. Pietrantoni shows a figure giving the very intimate connections between the tip of the petrous bone, the Gasserian ganglion and the sixth nerve and the petrous sinus. The abducent runs close along the tip of the petrous bone and is almost surrounded by a plexus of veins which come from the petrous bone tissue and empty into the inferior petrous sinus. Above and externally other vessels pass from the petrous bone to the Gasserian ganglion. These venous pathways, as I will show later, determine the direction, not only of congestion, but also of osteitis and suppuration. The surgical procedures must follow these pathways in creating drainage as well.

These routes described by Pietrantoni, however, are not yet complete, because we still have further communications of great significance, particularly in regard to the syndrome which I am describing.

There is a wide distribution of vessels that follow along with the middle meningeal artery, the venous elements of which return to the pterygoid plexus. By tracing the way of the middle meningeal artery we get the following interesting distribution. The middle meningeal artery, accompanied by the vein, passes through the foramen spinosum to enter the middle cranial fossa. After traveling a short distance in the groove on the great wing of the sphenoid, between the dura mater and the bone, it divides into anterior and posterior terminal branches having the following distribution:

1. Superficial Petrosal.—A small branch which arises from the middle meningeal soon after it enters the cranium. It passes through the hiatus canalis facialis and anastomoses with the stylomastoid branch of the posterior auricular artery; it also sends some small branches into the tympanic cavity.

2. Ganglionic.—Minute branches which supply the semilunar ganglion and roots of the fifth cerebral nerve.

3. Superior Tympanic.—A small twig which reaches the tympanic cavity through the canal for the tensor tympanic muscle or through the petrosquamous suture.

4. Orbital.—An anastomosing branch which arises occasionally from the anterior terminal branch. It passes through the superior

orbital fissure into the orbit and anastomoses with the lacrimal artery.

5. Anterior Terminal.—Spreading to the anterior and posterior cranial fossa, the largest of the two terminal branches, passes upwards along the great wing of the sphenoid to the sphenoidal angle of the parietal bone, where it is sometimes enclosed in a distinct bony canal.

6. Posterior terminal to the vertex and posterior cranial fossa.

7. The accessory meningeal branch which may arise directly from the internal maxillary or from the middle meningeal, passes upward on the medial side of the external pterygoid muscle and enters the middle fossa of the skull through the foramen ovale, supplies the semilunar ganglion and the dura mater, and terminates by anastomosing with branches of the middle meningeal and internal carotid arteries. The venous return of the same route empties into the pterygoid plexus. From this description it is seen that the venous drainage of the tympanic cavity passes not only to the petrosal sinuses and the cavernous, but also directly to the pterygoid. Congestion affecting the pterygoid plexus would also tend to manifest itself along the course of the veins emptying into the plexus. These tributaries supply the semilunar ganglion, the canal for the tensor tympanic, orbital and the temporal to the dura mater. Schwalbe, considering the venous drainage of the tympanic cavity, remarks that it has not been sufficiently studied. In general, they correspond to the arteries and drain partly into the middle meningeal vein, partly into the pterygoid plexus and partly (drum membrane mucosa) into the deep auricular vein.

In considering the reaction to inflammation occurring in otitis media where extension to the petrous bone is threatened, we are dealing primarily with an extraordinary degree of inflammatory reaction. This increase in severity also manifests itself quite late after a period of several weeks of otitis media, with venous stasis or after a prolonged otitis associated with mastoiditis. It is of interest that the evidence of extension to the petrous bone sets in almost collateral with the time of the mastoid operation and becomes evident usually at about the fourth or fifth postoperative day. At that time there appear signs of circulatory enbarrass-

ment, which either clear up spontaneously or after an interval of apparent accommodation show secondary signs of further disturbance in the form of localized osteitis which may go on to bone coalescence and suppuration remote from the tympanic cavity. The point of predilection for this involvement extending into the petrous bone is apparently the petrous tip or the groups of cells, chiefly the pericarotid or the sublabyrinthian spreading toward the tip. With involvement of the tip comes an additional group of symptoms. In studying the progress of inflammation in bone we find that following the period of venous congestion and stasis there occurs either resolution of the process or bone coalescence and suppuration. Since inflammation of the petrous bone confronts a fairly rigid system to inflammatory reaction with slight possibility for swelling, bone destruction results. In suppuration of the petrous bone it is still a matter for investigation whether the venous stasis does not predispose the tip to the coalescing processes remote from the original focus reported in the various cases in the literature. An additional factor tending to cause coalescence of the petrous tip is the sclerotic character of the shell of the petrous tip associated with pneumatization within. Outstanding in the entire consideration of the subject is the fact that the involvement of the petrous bone is most frequently, if not almost as a rule, secondary to pathologic processes in the middle ear rather than the mastoid process. The Gradenigo syndrome represents this variety of symptoms. From the study of the literature it is apparent that two outstanding types occur; one associated with suppuration of the petrous bone, which may go on to spontaneous evacuation or if not surgically relieved proceeds to intracranial complication; the other a state of simple continuation of the middle ear inflammation but with association of temporary involvement of the sixth cranial nerve and the semilunar ganglion. The former type leads frequently to intracranial complication, while the latter type usually resolves spontaneously. For the former type a variety of routes of extension of the suppuration from the middle ear or mastoid to the petrous tip have been considered. For the latter the phrase "toxic" is usually reserved.

In my studies I have found that the earliest involvement of the petrous bone manifests itself in the form of venous congestion

or stasis, directly demonstrable, first by its influence through the middle meningeal vein on the pterygoid plexus; later through the inferior petrosal sinus on the petrous tip. The symptoms of involvement of the middle meningeal vein and pterygoid plexus are a triad of (1) edema of the lower lid, (2) moderate or severe trismus with pain and difficulty of movement of the pterygoid and masseter muscle, and (3) temporomaxillary and orbital pain extending to a low grade major trigeminal neuralgia. These three symptoms I have observed and present here as a syndrome indicating venous congestion extending from the tympanic cavity to the petrous bone and preceding or homologous with the Gradenigo syndrome which represents a later stage of congestion of the petrous tip. The venous drainage of the anterior tympanic cavity is not as rich as that of the posterior part and mastoid, so that circulatory embarrassment of the anterior portion is more likely to occur early. It is only in the progressive cases that the venous congestion will manifest itself in the tributaries of the superior and inferior petrosal. The triad of symptoms which I have described are easily comprehended when one bears in mind the anatomic relationship which I have already described between the Gasserian ganglion, the dura of the temporal region and the congestion of the orbital veins leading to edema of the lower lid. As I have already indicated, these symptoms when they occur will usually clear up spontaneously with the accommodation to the circulation in the pterygoid plexus. The edema of the lower lid is usually attributed to a tight bandage, and the pain on movement of the jaw and the headache considered merely as a coincident to the otitis. These symptoms are, however, guideposts to the extent of involvement of the venous circulation. The direct relationship of these symptoms to the circulation of the middle ear is demonstrable by the very rapid improvement of the symptoms on incision of the drum membrane and the free bleeding induced from the middle ear. Pietrantoni, in his study of the Gradenigo syndrome, likewise has observed the relationship of the tympanic veins to that of the petrous bone involvement and has suggested inducing free bleeding from the middle ear as a method of rapidly reducing venous congestion of the tip of the petrous bone.

In addition to presenting here these cases of my own, I am able to show from the literature case reports where, on empirical grounds, incision of the drum membrane and free drainage from the middle ear exercised a beneficial influence on cases of involvement of the petrous bone.

This presentation of the mechanism of involvement of the petrous bone tends to clarify our conceptions regarding the Gradenigo syndrome or, as it is also called, the syndrome of the petrous tip. Gradenigo has described the propagation of inflammatory processes from the tympanic cavity to the petrous tip, manifested by evidence of involvement of the sixth cranial nerve and the Gasserian ganglion. These organs may be involved simultaneously or independently. In the latter case it appears as a trigeminal neuralgia.

In viewing the Gradenigo syndrome, we see a state of involvement of the petrous bone greater than that in the syndrome which I here described. Here as we approach the petrous tip we find as one of the early signs involvement of the sixth cranial nerve. The sixth nerve palsy that is encountered is usually a temporary symptom which, almost as a rule, clears up unless signs of intracranial complications develop. The evanescent character of this palsy as well as the relief from the Gasserian ganglion symptoms are usually ascribed to some vague toxic factor, but can be more definitely shown to be the result of venous congestion passing centrally from the petrosal sinuses. The most probable site where the venous engorgement affects the sixth cranial nerve is in the canal of Dorello. Here the sixth nerve and inferior petrosal sinus pass through a short osteofibrous narrow canal (Dorello canal). Enlargement of the inferior petrosal sinus can only occur at the expense of the sixth nerve, and pressure here would cause a distinct interference with the nerve function until circulatory compensation has occurred and nerve recovery allowed. This contention has been advanced by Dorello and was disputed on the ground that the veins of the tympanic cavity which could explain the diffusion to the tip were not tributaries of the inferior petrosal sinus. This, however, has been refuted by the work of Pietrantoni who has demonstrated the drainage from the tympanic cavity to the inferior petrosal sinus. It has also been maintained

that the temporary compression of the abducent in the canal of Dorello would not account for a palsy that may last several weeks. That this is not so can readily be realized when one considers the duration of the facial paralysis in Bell's palsy or in the mastoid postoperative cases where temporary pressure on the nerve is followed by prolonged palsy.

In accepting this venous aspect of involvement of the petrous bone we are able to explain more readily the simultaneous involvement of the abducent nerve and Gasserian ganglion. The fact that the petrous tip is frequently pneumatized, as can be seen from my demonstrations, does not imply that there is therefore involvement of the Gasserian ganglion and the abducent nerve. At the petrous tip the cell formation is usually encased in heavy solid bone, the erosion of which would imply an intracranial complication. There is no doubt that such complications do occur, as has been shown by Eagleton, Friesner, Kopetzky and others, but those are not to be listed as cases of Gradenigo syndrome, but rather complications of those cases.

It can be seen from this discussion that the syndrome of Gradenigo really represents one referring to venous disturbances rather than to suppuration, and when restricted to this anatomic pathologic conception presents a definite picture. However, as can readily be understood, this venous process when not relieved will pass over to a suppurative state and at autopsy show definite suppuration at the tip, thus leading earlier observers to conclude that the sixth nerve palsy and Gasserian neuralgia were secondary to suppuration of the petrous tip. It is the object of this paper to point out, first, that the Gradenigo syndrome is a venous manifestation and not necessarily associated with a suppurative process. This also explains the high percentage of spontaneous recovery in cases evincing the Gradenigo syndrome; second, that a similar syndrome appears earlier in some cases, which I am describing as associated with the anterior venous drainage and characterized by circulatory signs on the part of the middle meningeal vein with temporomaxillary dural pains and edema of the lower lids from stasis in the pterygoid plexus and inferior orbital veins, as well as trismus of the pterygoid and masseter muscles. Both of these syndromes are essentially not signs of suppuration

but rather premonitory of suppuration. I am also pointing out that the induction of free bleeding from the tympanic cavity tends to offer some relief to this venous disturbance and is followed by prompt improvement in the symptoms. This is likewise suggested by Pietrantoni for the Gradenigo type.

Kopetzky and Almour also point out that suppuration of the petrous tip may occur without the appearance of the Gradenigo syndrome and, in fact, suggest that the presence of abducent palsy is more apt to point against than to the diagnosis of petrosal tip suppuration. This objection, however, is explained by the introduction of my observations showing that an anterior venous syndrome can occur without the Gradenigo syndrome and indicating early involvement of the petrous bone, along an anterior route. This I later point out was evident from Kopetzky and Almour's own cases but not recognized by them.

In considering the Gradenigo syndrome, it is well to inspect both his own original publications as well as those of his staunch supporters, Citelli and Lasagna.

It becomes apparent that involvement of the petrous bone gives risé to a variety of mechanisms, one of which brings with it paralysis of the sixth nerve, the other an absence of such involvement. The former usually runs a mild course, more frequently with recovery; the latter pursues a more dangerous path. This difference has led to acute controversy in every discussion of the Gradenigo syndrome. Kopetzky is of the opinion that the abducent paralysis leads away from the diagnosis of suppuration of the petrous tip, because most of his cases of petrous tip suppuration showed no abducent paralysis. Yet the literature reveals numerous cases where abducent paralysis preceded fatal intracranial complications with demonstrable necrosis of the petrous apex.

A searching investigation of the detailed anatomy brings with it important light on this perplexing situation. The two types of clinical pictures, those with and those without abducent paralysis, really represent two separate pathways to the petrous apex: one a posterior route, such as is represented by the posterior perilabyrinthine cells, spreading to the tip, and the hypotympanic group of cells; the other route is the anterior perilabyrinthine

and pericarotid group spreading to the petrous tip. These groups have been painstakingly demonstrated in my radiographic illustrations, which will be presented later. The posterior group when involved occurs by way of the tympanic veins towards the inferior petrosal sinus. Stasis in this group would lead to pressure on the abducent in the canal of Dorello, as described by Dorello and substantiated by Pietrantoni. Thus we would see at first diplopia. Should actual osteitis and suppuration of the cells in this pathway occur we would find suppuration of the petrous tip with coalescence of the cells. Such destruction would also be more directly reached by the hypotympanic route, which I shall later describe.

In sharp contrast to this is involvement along the anterior group of veins. Here we would be more likely to see in the very early stages the syndrome which I have described earlier of edema of the lower lid, trismus and temporomaxillary and orbital pain, representing stasis of the veins draining through the middle meningeal vein and pterygoid plexus. In the absence of the observation of this syndrome progression of the disease with involvement of the anterior perilabyrinthine and pericarotid cells would lead to coalescence of the petrous tip. In this latter course there would be no mechanism to involve the inferior petrosal sinus or the canal of Dorello and no abducent paralysis. Surgical drainage of this path would necessarily follow the line of invasion and be amenable to drainage by the technic described by Almour. This technic would not, however, be so applicable to the posterior type of invasion which I have described. The pathologic anatomy, therefore, guides our surgical approach.

With these conceptions before us, it is well to look again at further clinical data. It is enlightening to see the original report of Gradenigo, translated literally from the Italian, on "Paralysis of the Abducent of Otitic Origin" (194):

"At the sitting of January 29, April 22 and June 17, u. s. of the Royal Academy of Medicine of Turin, I established the existence of a particular pathologic syndrome characterized by acute otitis media, by very intense and tenacious pains in the affected ear and in the corresponding half of the head and by the appearance, after a period of time varying usually from three weeks to one and one-half months from the inception of the otitis, of a paresis or isolated paralysis of the right external ocular on the side of the affected ear. I have had occasion to observe and to publish six cases of such an affection of which five recovered completely and

one ended in death by the arising of diffused purulent leptomeningitis. In all probability it is a question in such a malady of a circumscribed focus of leptomeningitis, whether purulent or simply serous, due to infection spread to the meninges from the tympanic cavity, this meningitic focus is cured for the most part with the healing of the temporal bone, but, in some cases is succeeded by generalized meningitis. Scattered through otologic literature I have found only four cases which correspond perfectly to the syndrome described by me which we owe to Spira[1], Pischel[2], D'Ajutolo[3] and Torok[4], such rarity is probably only apparent and due to the fact that being ordinarily benign such cases are not deemed worthy of publication[5]. In this opinion, I am confirmed by the fact that after I had called attention to the pathologic syndrome in question, three distinguished Italian otologists, my colleagues Ricci of Treviso, Mongardi of Bologna, and Citelli of Catania, let me know that they too had observed a case of the affection and were so courteous as to send me the respective clinical histories which my readers will find set forth hereafter in detail.

At the meeting of the French Society of Otology held in Paris in May, four other cases of such an affection were reported, one by Lannois and Ferrand, two by Lubet-Barbon and one by Cheval. When these new cases are compared with those already published by me and with the few existing in the literature, one is struck by the perfect identity of the symptoms and of the clinical course. Thus we have to do with a well defined clinical syndrome, with constant symptomatology. The prevailing otitic symptoms are always connected with the infection of the tympanic cavities in the strict sense rather than of the mastoid cavities; when the severity of the painful symptoms led to operative intervention on the mastoid, the result of the exploration of the antrum and of the cells was often almost negative; thus, also the absence of manifest lesions of the floor of the fossa crania media and of the external face of the dura mater which could sometimes be recognized by an explorative craniotomy. In some cases the infective process of the tympanic cavity was clearly localized in the epitympanum. The infection probably spreads from the cavity to the meninges; this opinion is supported in two of my cases by the existence of symptoms of serous leptomeningitis, respectively purulenta diffusa. Besides in a later case observed by me, which however, does not enter the clinical picture traced, because the ocular paralysis was connected, instead of with acute otitis, with chronic purulent otitis with cholesteatoma, said paralysis had supervened immediately after an operative intervention in the tympanic cavity, executed by a surgeon, an intervention which had provoked also lesions of the facial and of the labyrinth; this case demonstrates the possibility that infective lesions of the tympanic cavity spread to the base of the fossa cranii media, determining the isolated paralysis of the abducent nerve."

Gradenigo later, in 1908, again reported on "Paralysis of the Abducens in Cases of Otitis," and attempted to clarify the cases.

"Three years ago at several sittings of the Royal Academy of Medicine of Turin I called attention to a pathologic syndrome which I then had repeated occasions to observe, characterized essentially by the three following symptoms; purulent otitis media for the most part acute, particu-

larly violent and continuous pains in the corresponding parietal and temporal region, paralysis of the sixth nerve on the side of the diseased ear. At that time I had been able to find some analogous cases, scattered through the literature. Those publications of mine gave occasion thenceforth to numerous publications on the argument which succeeded each other rapidly, so that today I have been able to gather and study 57 cases of the syndrome in question (my own included), of which 22 belong to Italian authors. They are summarized with their most important particulars in the annexed tables.

I deem it opportune to tell briefly of the principal results furnished by such new material of observation, results which confirm essentially what I expounded in 1904 and enlarge our acquaintance with the mechanism of production of this particular pathologic form.

The cases hitherto observed allow themselves to be grouped into three categories. The first comprises the typical cases[24] in which is found only the symptomatic triad now pointed out; in the second group of cases with the characteristic phenomenology is associated some accessory symptom, in relation for the most part with irritative phenomena of the sixth or of the third nerve, or with meningitic irritation, or else complications are observed, like reactive mastoiditis, extradural perisinus abscesses, circumscribed, however, about the sinus sigmoideus, etc. The second group comprises more than half of the cases[29]. The tympanic perforation has its seat, with a certain frequency in the anterior segment of the membrane. Finally, in a third group can be placed the cases ending in death; the latter are fortunately rare (4 out of 57); death followed by purulent diffused leptomeningitis, with localization also spinal.

The anatomical pathologic substratum of the typical syndrome is given from the diffusion, favored by causes of retention of the pus, of the purulent infection from the tympanic cavity to the tip of the pyramid across the peritubal pneumatic cells and the canalis caroticus. Special attention was called to these cells, above all by V. Troeltsch, by Urbantschitsch, Siebenman and by Bezold, and, they were studied by Tonietti also. The abducent nerve is injured in relationship to the tip of the pyramid, just as it traversed the dura mater; so it is a question of a circumscribed osteitis of the tip of the petrous bone with secondary pachymeningitis and according to the progression circumscribed leptomeningitis; however, the fact that the injury usually maintains itself extradural explains the ordinary benignity of the affection. In some cases, by diffusion of the infection, there results purulent leptomeningitis at first limited then diffused.

The facts observed do not confirm the hypothesis advanced, among others, by Bonnier[24] and by Geronzi[10 41] of a vestibular reflex origin of the paralysis; indeed every symptom of labyrinthine injury is lacking. The hypothesis put forth by Dorello[62] of a compression of the sixth nerve in its course across the osteofibrous petrosphenoidal triangular space, has no clinical observation to support it.

Besides it is necessary to consider that the veins of the inflamed middle ear are not tributaries of the inferior petrosal sinus and that in the typical syndrome the labyrinth which pours its venous blood into the inferior petrosal sinus, does not take part in the pathologic process.

Not all paralyses of the abducent nerve in cases of otitis come under the clinical syndrome in question: Otitic paralyses of the abducent may

be due to various other pathogenic mechanisms, among which the most frequent are: deep extradural abscess of the upper border of the pyramid, as in cases of Citelli, of Poli, of Strazza, diffused osteomyelitis of the pneumatic structure of the temporal bone, which extends to the tip of the pyramid, as in a case recently observed by me.

Such cases are not naturally a part of the typical pathologic syndrome and have in comparison with the latter, different prognosis and treatment.

The treatment in the typical cases consists above all in favoring the drainage of the pus from the cavity through the natural or artificially executed or enlarged opening of the membrane."

These original reports of Gradenigo are not accompanied either by autopsy descriptions or anatomic demonstrations, and his assumption that he was dealing with a localized leptomeningitis is not substantiated. Pietrantoni's work has shown that the venous drainage also differs from Gradenigo's conception and tends rather to support Dorello's contentions.

In the light of these reports, it is well to look upon the Gradenigo syndrome as an indication of venous disturbance rather than that of suppuration of the petrous tip—an indication, however, that may progress to osteitis and suppuration. Also, that this syndrome has as its homologue a syndrome of similar type affecting the anterior group of veins, such as I have described in this paper. My own cases, which I am here reporting, tend to indicate the analogous character of the symptoms.

The first case is that of a boy of 7, M. F., who came to me in 1926 with a history of four weeks' aural discharge and a low grade fever ranging from 99 to 101. Examination revealed an anterior perforation with a rather profuse purulent discharge. X-ray examination showed a diffuse clouding of the mastoid process but no necrosis and abscess formation. He was hospitalized at the New York Post Graduate, where during a period of two weeks' observation the discharge continued with a persistent low grade fever. The child was apparently comfortable with very little complaint. The drum membrane, although bulged considerably, was relatively insensitive. There developed gradually sinking of the posterior canal wall and evidence by X-ray of bone coalescence. I performed a simple mastoid operation at which the coalescent type of mastoiditis was disclosed. Pneumatization was extensive, particularly towards the zygoma. After a two weeks' stay at the hospital the child was discharged for ambulatory care. The middle ear was dry, but the wound was discharging moderately. After the second ambulatory dressing there developed a definite edema of the left lower eyelid. He also began to complain of discomfort in the left side of the face and the infra-temporal region. During the next week he developed a slight abducent paresis of the left side and some orbital pain. The symptoms subsided after paracentesis.

The second case is that of J. M., a young man of 19, who began with severe right otitis media, showing hemorrhagic bullæ in the drum membrane and associated with profuse serosanguinous discharge. During the third week of the otitis he showed a definite mastoiditis with extensive pneumatization of the petrous bone. There was slight clouding apparently of the entire petrous bone. He was admitted to the Park West Hospital April 14, 1929. The next day mastoidectomy was performed, following which the symptoms all cleared and the patient was comfortable until the third day, when there was a slight rise in temperature and a recurrence of the edema of the right lower eyelid; in addition there was again temporomaxillary and orbital pain and moderate degree of trismus. Incision and free bleeding from the middle ear brought about almost immediate relief from the symptoms and prompt lowering of the fever.

The third case was that of Miss F. M., a young woman of 28, who showed that on the third day postoperative to a mastoidectomy a moderate edema of the lower lid in the same side and temporo-maxillary discomfort. This disappeared spontaneously the next day.

Asherson, in the Journal of Laryngology, page 827, remarks that edema of the eyelid, in association with acute mastoiditis, is a phenomenon that has received scant attention at the hands of otologists. The edema is unilateral and limited to the lower eyelid on the same side as the diseased mastoid. This physical sign is consistently detectable in children in association with a certain type of acute mastoiditis. He recites the case of a child of 7 with a subperiosteal abscess and associated unilateral edema of the lower lid on the same side. This edema passed off in a few days after mastoidectomy. S. M. Smith (Jackson and Coates, "Diseases of Throat, Nose and Ear," page 528) refers briefly to slight edema of the eyelids in zygomatic mastoiditis. W. M. Mollison ("Paralysis of Eye Muscles Occurring in Connection With Mastoiditis," British Journal of Children's Diseases, Vol. XVIII, No. 18, 1921) mentions edema of the side of the face and eyelids following a radical mastoid in a child aged 12. Again, in 1922, he refers to edema of the eyelid in a case of zygomatic mastoiditis in a child of 12. Horgan also reports homolateral edema of the eyelids in two cases, aged 14 and 15, respectively, of delayed zygomatic mastoiditis, both as a sequel of a mastoid operation.

No direct explanation is attempted by any of these observers except Asherson. He suggests that the edema is due to pressure on the veins returning from the outer canthus of the eye by the temporal fascia. This explanation has no serious anatomic basis.

He also suggests the possibility of venous obstruction of the scalp veins causing the edema of the lower lid, and also the possibility of stasis being transmitted by the superior petrosal to the cavernous sinus. The fact, however, that in my cases the edema of the lower lid was directly relieved by incision of the drum membrane and free bleeding from the tympanic cavity, tends to bear out my explanation for this phenomenon. In support of my contentions are the case reports of Neumann where on the purely empiric basis circulatory relief followed incision of the drum membrane after mastoidectomy.

Schlander has reported two cases from the Neumann clinic in Vienna.

The first case was that of a young lady of 29 who came to operation because of a three week otitis which had from the onset been marked by severe neuralgic pains in the occiput and vertex. At operation there was a perfectly pneumatized large mastoid in the cells of which was stringy pus and swollen mucosa. The cells extended deep into the zygomatic process and widely towards the bulb. In the antrum there was pus under pressure and granulations. Nothing was exposed. After the operation, on account of infection of the soft tissues, there was high fever. Eight days after the operation the patient began complaining for the first time of pains in the eye on the same side which she described as severe, stabbing and shooting, and also pains in the temple of that side. The pains increased particularly in the evening and at night the temperature stayed subfebrile.

Twelve days after the operation there appeared for the first time a change in the appearance of the drum membrane in the sense that now it was thickened and bulging, although on the fifth day postoperative it had already been pale.

One day later, that is, on the 13th day postoperative, a paracentesis was performed. Pus emptied itself under pressure and was followed by the extrusion of a granulation. Two days after the paracentesis, that is, on the 15th day postoperative, the patient was for the first time free from the pains in the eye and temple. From this day on she felt well.

On the fourteenth day after the paracentesis, that is, on the 27th day after the operation, a new complication appeared in that accompanied by vomiting and disturbances in equilibrium, a typical fistula symptom appeared which could be elicited as well by compression of the air in the external auditory canal as by pressure through the tampon in the retro-auricular wound.

Since the patient stayed free from fever and the labyrinth disturbance and fistula symptom disappeared during the subsequent eight days, no further intervention was attempted and the patient was discharged. The retro-auricular wound was closed and the canal dry. On the drum was a pin head sized granulation.

The second case is that of a 7-year-old patient, who had to have an antrotomy done because of a three weeks' duration otitis media. At opera-

tion there was found a large empyema of the mastoid which extended to the dura of the middle fossa and deep into the cells of the zygomatic process. The postoperative course was undisturbed and afebrile for thirteen days.

On the thirteenth day postoperative the temperature rose to 37.7 associated with vomiting. At the same time severe pains in the right ear and right eye set in with attacks marked by short or longer intervals. The pains radiated into the right temple and towards the left. The patient kept the right eye continuously closed. There was neither abducent paresis nor double vision. When asked why he kept the right eye closed he repeatedly replied "it hurts me." Since the pains associated with a subfebrile temperature continued, and the X-ray report (E. G. Mayer) showed a high grade atrophy of the petrous tip, hazy cortex and a central homogeneous increase in illumination; because of the threatening clinical picture and the disturbing X-ray findings, operative exposure of the pyramidal tip was considered.

Since simultaneous with the onset of these subjective tip symptoms, there had set in a new reddening and swellings of the hitherto reactionless drum a wide paracentesis which evacuated a profuse amount of mucopurulent discharge from the middle ear was done on the sixth day of the onset of the symptoms. On the second day after the paracentesis the eye pains were barely present and from the third day on the patient continued to be entirely free from disturbance.

Both of these reports demonstrate cases of acute otitis, both of which after mastoidectomy developed symptoms characteristic of involvement of the petrous bone, yet were definitely relieved by paracentesis. This, to my mind, was due to relief of venous congestion which promptly relieved the symptoms. I feel that it is important to give proper emphasis to this point. Because of the growing interest in surgical interference on the pyramidal tip it is possible that the proper and adequate conservatism may be set aside for more ready surgical intervention. Certainly in the latter case there was ample inducement for surgical attack to the less conservative minded.

Since this paper is not intended to include a study of actual suppuration of the petrous bone, but rather to point out the rôle of the venous system in causing symptomatology which is frequently attributed to suppuration, I am not here going into the differential diagnosis of suppuration of the petrous pyramid. The recognition, however, of such circulatory embarrassment of the petrous bone must, however, be considered a serious sign and construed as a forerunner of osseous involvement. The surgical indications in the early stages are for wide paracentesis and the induction of free bleeding from the middle ear.

In line with these conceptions is the recent report of Busch in his paper on "Nonfebrile Sinus Thrombosis With Paralysis of the Abducent" (Zeitsch. f. Laryng., Rhin. [Tril. 1: folia otolaryng.], 21:165, June, 1931). He reports that his case showed (1) no fever and hardly any symptoms; (2) the abducens became involved. That cases of sinus thrombosis develop entirely without or with only slight symptoms is a rare, but not unknown occurrence. In regard to the second unusual aspect, the paralysis of the abducent nerve, the author, after discussing various possibilities of pathogenesis, comes to the conclusion that in this case the sinus thrombosis was the only (mechanical) cause of the involvement of the abducens nerve, for following the operative removal of the thrombus the paralysis disappeared. This involvement occurred by way of the inferior petrosal sinus.

Another significant point in the study of embarrassment of the venous bed of the petrous bone is the relative frequence of the onset of these symptoms after a week or more following the mastoid operation. The tympanic membrane tends to close, thus making conditions for the relief of congestion in the tympanic cavity less favorable than hitherto. It is, therefore, important to watch for the early signs, such as I have described in detail in this paper, and to think promptly of supplying and maintaining free drainage for the middle ear. Obstructive granulations in the middle ear can be observed and are repeatedly reported in cases of either Gradenigo syndrome or petrous suppuration (Wesley C. Bower's case—Laryngoscope). It is extremely significant to observe that almost as a rule cases showing petrous bone involvement also show granulations in the middle ear. The granulations in themselves indicate an inflammatory process deeper than the mucosa. This also is the probable seat of minute thrombophlebitis of the smaller veins which create the mechanism for venous stasis and is the forerunner of osteitis.

It is well also to point out here that the relationship which the middle ear bears to the petrous bone is similar in character to that which it bears to the mastoid process. However, the relative smoothness of the median tympanic wall and the interposition of the hard bony labyrinth acts as a barrier to invasion, whereas

the ready accessibility of the mastoid pneumatic cells makes them the outstanding avenue for invasion.

The great importance of watching the venous pathways of the temporal bone is graphically shown in the case reported by Richard T. Atkins. It also demonstrates the importance of a carefully taken history and the significance of the interpretation.

The case is that of a male, age 7 years. Admitted to the Harbor Hospital, April 5th, 1927. Eleven days prior to admission the patient developed an acute otitis media on the left side, following an acute nasopharyngitis. The drum membrane was promptly incised. The ear drained profusely and there was moderate elevation of temperature. Three days after the onset, a painful swelling developed in front of the ear. On admission to the hospital, the temperature was 101.4 F., pulse 120, and respiration 28. There was a marked tender edematous swelling in front of the ear, which extended up into the scalp. Movements of the jaw were painful and markedly limited. There was mastoid tenderness; profuse purulent discharge; and narrowing of the canal at the fundus. The leucocyte count was 21,300, of which 76 per cent were polymorphonuclears. A simple mastoidectomy was performed. The cells were of the large pneumatic type. They were generally filled with exudate and there was free pus in the tip and suprasinus region. There was a large cellular zygomatic recess which was filled with pus, and, at the junction of the middle and posterior roots of the zygoma, a perforation was found which led into a localized abscess, from which an ounce of pus was evacuated. No dura or sinus was exposed. A counter opening was made in front of the ear and a rubber dam drain inserted. Culture of the pus from the mastoid showed a growth of hemolytic streptococci.

Following the operation the swelling completely subsided; the patient was apparently making a good recovery, until on the tenth day following the operation, he began to complain of pain the left eye. The pain continued for the next few days, and, occasionally he also complained "of pain in the teeth and the left side of the face." The temperature oscillated between normal and 101 F. The leucocyte count was 26,500, of which 77 per cent were polymorphonuclears. The urine contained a faint trace of albumin, a few hyaline, granular and mixed casts, and, at times there were a few pus casts. The eye grounds were negative.

Later, three weeks following the mastoid operation, the patient complained of double vision, in addition to the pain, and, soon afterward developed a paralysis of the left sixth nerve. At this time there was also a sudden rise of temperature to 106 F., which was not preceded by a chill. The patient was nauseated, but, did not vomit.

The leucocyte count was 11,000, of which 62 per cent were polymorphonuclears. Culture of the blood was negative. The cochlea and semicircular canals were active. The middle ears, which had previously been dry, now began to discharge again. Because of the temperature reaction and the sixth nerve paralysis, an exploratory operation was performed on the lateral sinus. The internal jugular vein was ligated, and, the lateral sinus opened and packed. There was no demonstrable clot found. Following the operation the temperature dropped to 101 F., and the patient

seemed more comfortable. The pain was less severe and the paralysis less marked.

Later, however, the pain returned, and the patient also complained of some pain in the right side of the face and right eye. He had restless nights, due to the pain and headache. There was some pain and numbness of the legs. The temperature ranged between 100 and 103 F. Culture of the blood still remained negative. The leucocyte count was 25,800, of which 70 per cent were polymorphonuclears. As it was thought at this time that the patient had an epidural collection of pus in the middle fossa, another exploratory operation was performed. This consisted of an exposure of the dura of the middle and also the posterior fossae. There was no pathology found. The patient was being transfused every other day, with no apparent beneficial effect. Neurologic examination by Dr. Foster Kennedy revealed the following: Pupils brisk, discs clear, no swelling. No V or VI palsy. Kernig's sign present R/1. All reflexes normal. No sign of brain abscess. Meningitis is of dura and quite well localized. Leucocyte count 30,000, 82 per cent polymorphonuclears. The temperature now remained fairly high, 102-105 F. The patient complained of pain in the neck and back. He was irritable and very restless. Later, Dr. Kennedy reported as follows: Kernig more marked. No sign of brain abscess. There is threat against the meninges, but all present symptoms are compatible with meningismus and sinus thrombosis. Another operation was performed by Dr. Alfred S. Taylor, one month after primary operation. The posterior and middle fossae were explored, but no collection of pus was found. The next morning the patient suddenly became cyanotic, lapsed into coma, and died. Autopsy revealed: An extradural abscess over the apex of the petrous pyramid had evidently ruptured and entered the basal cistern, finding its way from the point through the leptomeningeal spaces. The primary process seems, therefore, to have been the extradural abscess, associated evidently with an osteomyelitis of the petrous bone. Anatomic Diognosis: Osteomyelitis of the petrous bone. Extradural abscess. Suppurative cerebral leptomeningitis. Thrombophlebitis of the inferior petrosal sinus.

On reviewing this case, one sees first that shortly after the onset of the otitis media there developed a painful swelling in front of the ear, associated with painful movement of the jaw and some limitation of motion. Whether some edema of the lower lid was observed was not recorded. It is, however, suggestive that venous pathways from the tympanic cavity affected in this case are the anterior ones, draining into the pterygoid plexus and associated with trismus. With this was apparently suppuration of the zygomatic cells. This anterior involvement makes invasion of the petrous bone much more likely, because here, instead of the protective barrier of the labyrinth bone, we have direct cell groups extending toward the petrous tip. Ten days after operation evidence of involvement of the petrous bone is beginning

to appear in the form of pain in the left eye. This is more suggestive of congestion of the posterior group of veins. On the third week, eleven days after these initial symptoms, came signs of abducent paralysis, indicating progression of the process at the petrous tip. The temperature rose to 106, followed by recurrence of discharge from the middle ear, thus indicating the character of the invasion. Incision of the lateral sinus and ligation of the jugular afforded transient relief in the symptoms, probably from the temporary improvement in the venous drainage of the petrous bone. Another operation to expose the dura of the middle fossa also revealed no pathology. The fourth operation exploring the posterior and middle fossa also revealed no pus.

The autopsy of this case bears out the anatomic thesis presented in this paper. The primary signs of invasion of the petrous bone were venous on the part of the anterior group followed subsequently by involvement of the posterior venous mechanism with a typical Gradenigo syndrome, which is proven by the finding at autopsy of thrombosis of the inferior petrosal sinus without involvement of the lateral sinus. Progression of these syndromes into the suppurative state thus leading into necrosis of the petrous tip with terminal meningitis.

I have described this case in great detail to indicate that the surgical approach to the diseased area must be followed, not by choosing standard methods of exposure, but rather by determining which of the venous groups have led the invasion into the petrous bone. I have pointed out that there are five major paths, four of which Pietrantoni has emphasized.

First—The small vessels of the tympanic cavity to the pericarotid plexus towards the cavernous sinus (anterior group). Second—From the tympanic veins to the inferior petrosal sinus (posterior group). Third—From the tympanic veins to the superior petrosal sinus (posterior group). Fourth—Following the cell groupings, peritubal and perilabyrinthine, towards the tip. Fifth—The anterior tympanic veins that drain into the pterygoid plexus by way of the middle meningeal veins.

It becomes obvious, therefore, that no one surgical approach to the petrous tip can suffice for all cases, nor can any one method be advocated too rigorously. From my anatomic and roengeno-

logic studies one can see that although the route suggested by Almour would be excellent for the anterior group, it would not be advantageous for the posterior or inferior group. Here, as in the cases of radical frontal sinus operations, the choice of type of operation is dictated by the type of pneumatization to be dealt with. The guide in these cases of petrous bone involvement is primarily the history to determine whether the invasion is anterior, as manifested by the triad of symptoms which I suggest, or posterior, as manifested by the triad of symptoms suggested by Gradenigo. The second factor in helping determine the route is the study of the X-ray.

Viewed from this angle, many of the differences in opinion regarding the evaluation of the Gradenigo syndrome are better understood. Those studying cases of petrous bone suppuration developing along the anterior route might have no signs of abducent paralysis, while those progressing along the posterior route would be more likely to have the Gradenigo symptoms.

In the series of nine cases of suppuration of the petrous bone presented by Kopetzky and Almour, two showed premonitory symptoms of venous congestion, both of which were of the anterior group which I have described in this paper. In his series, case three, it was manifest at the first examination that there was pain and swelling of the right eye, and, in case six, in a bilateral mastoidectomy, swelling of the right eye manifested itself directly after operation. Both cases subsequently developed into a petrous bone suppuration. Case three of their series is as follows:

"Case 3.—L. S., a girl of 12 years, was first seen on April 7, 1930. Three weeks before, she had caught cold, and two weeks later she developed pain in the right ear. Paracentesis was followed by a thick yellowish discharge. At the first examination she was complaining of pain over the right eye, which was swollen. Both physical and radiographic examination revealed mastoid involvement. Mastoidectomy was performed that day. The mastoid was exenterated to the inner table, exposing the lateral sinus near the knee and lower down toward the posterior meatal wall. The dura was exposed near the zygoma. A large development of the zygoma was found with infected diploic tissue and cells extending far forward into the root of the zygoma; cellular elements were also found behind the horizontal semicircular canal, and downward into the petrous pyramid. These purulent tracts were followed into the depth of the pyramid, and the mastoid tip was also removed. The wound was closed with dermal sutures. A rubber drain was left in situ. The immediate reaction to operation was entirely usual. On the following day the tem-

perature rose from 99.4 to 103.8, but in the next few days it gradually came down. The child was restless and continued to complain of pain in the ear. Two days after operation she began to complain of slight abdominal pain but, although the abdomen was slightly distended, there were no neurologic signs at that time.

On April 12th, the fifth day after operation, on which the highest temperature was 101, a full dressing was done and all the stitches removed. There was some stitch abscess; and an edema was present about the posterior scalp edge of the wound. Pus was expressed and the surface painted with iodine and alcohol and cleansed. The middle ear was still discharging, and the child still complained of retro-orbital pain on the right side in occasional spasms. On April 13th the temperature ranged between 99.6 and 101.6; on the 14th the range was between 99.2 and 101.6. The pain in the eye was less and there was no longer a complaint of cramps. For the next few days the temperature range was not great—between 99.2 and 100.2 on the 15th, between 99.8 and 100.2 on the 16th, and between 99.2 and 101 on the 17th. On the latter date the retro-orbital pain was almost gone. On dressing the wound adaptation was found to be satisfactory, and the amount of pus materially less. There was still a considerable middle ear discharge. On April 17th the patient began to complain of pain in the back of the right leg and of severe pain in the right eye, which appeared more swollen. She was more drowsy. The following day the complaint was of pain in the eye and ear; the patient did not feel well, and there was a sharp rise of temperature from 98.8 to 103. The discharge from both the middle ear and the mastoid was less purulent, and the appearance of the wound was satisfactory. The following day, April 19th, the temperature continued septic (from 99.4 to 102.8), the blood picture was about the same. A consultation was held, and in view of the septic type of the temperature and persistent pain in the right eye, reoperation was decided upon. The old wound was laid open, the lateral sinus was examined and was cleared of a thrombus and the internal jugular was ligated. There was a sharp temperature reaction the following day, from 100 to 104.2, the patient complained of slight frontal headache over the eyes and of pain on swallowing. There was some hoarseness, but the larynx was found to be of normal motility. She was restless that evening, changing her position frequently and sitting up to do so. The next day the child was very restless and irritable, changing her position frequently. She complained of pain in the ear, difficulty in swallowing and pain over both eyes, at times in the right eye alone. On April 22nd there was less pain in the ear but she still complained of pain in the eyes, particularly the right and there was a recurrence of the pain in the stomach. That afternoon she began to complain of dizziness when turning from side to side. The temperature was between 101 and 101.8. On the following day the temperature range was between 100.6 and 102. The patient was still restless and continued to complain of pain in the ear and eyes, chiefly the right, and of dizziness on turning. A slight horizontal nystagmus was observed when the eyes were turned to the extreme right. When the C fork was placed on the forehead it was heard in the diseased ear. When the noise apparatus was placed in the left ear, she heard the spoken voice in the right. She did not lie in 'Forced position.' A full dressing was done, and all gauze removed except the plugs in the sinus. Then the ear was washed with Dakin's

solution and the patient complained of dizziness. A rubber dam was inserted and the neck wound dressed. The following day the child complained a great deal of pain in the right eye, and both eyes appeared slightly swollen, perhaps due to crying. She continued to complain of pain in the ear and abdominal cramps. She was nauseated at times, but there was no actual complaint of dizziness. The temperature ranged between 99 and 101. On April 25th the temperature ranged between 99 and 101.4, the child continued to complain a great deal of pain in the right eye and both eyes were swollen. She complained of dizziness in the morning and was drowsy. The following day the child was drowsy. When she was awake she complained of feeling tired most of the time, and was restless. She had severe pain in the right eye and the eye and cheek were swollen. The temperature was between 99.6 and 101.6. The next morning, April 27th, there was a sharp rise in temperature, from 100.4 to 104.8. The child continued tired and the pain in the right eye was severe. At times both eyes were complained of. Her memory seemed poor and she began to jerk her leg (the nurse could not determine which, because of her position) and jerkily flex both hands towards her arms; this was observed chiefly on the right side. She was nauseated, but did not vomit. There was a spontaneous nystagmus when she shifted position, yet she continued to lateralize sound to the diseased ear, and to hear sounds with the noise apparatus in the healthy ear. A full dressing was done and no pus found either in the neck wound or when washing through the middle ear. Neurologic examination was negative except for a slight Kernig and a blurring of the optic disc on the nasal side. X-ray of the right petrosal pyramid revealed a blurring of its outline at the tip. A spinal tap was done and 5 c.c. of clear fluid removed under great pressure. The report revealed the fluid to be slightly cloudy but sterile; albumin was present, there was a faint trace of globulin; two white cells were found and a moderate number of red.

On April 28th the temperature continued to range high, between 101.8 and 104.4; the pain persisted in both eyes; and there was a nystagmus in both horizontal planes in addition to a slight vertical rotary nystagmus; she still heard sounds in the affected ear. Neurologic examination revealed a low grade swelling of the optic discs with blurring of the margins and an increased filling of the veins. There was a right Kernig. The deep reflexes were equal. The left plantar was equivocal. Weber was to the right. In view of the persistent pain in the eye, the symptoms of intracranial involvement, the septic temperature and the radiographic corroboration, operation was undertaken that day. The packing was removed from the mastoid cavity, and the wound opened wide. The sinus was found obliterated and the tegmen above the knee of the sinus was curetted. The mastoid was converted into a radical cavity. There was extensive cellularization of the entire root of the zygoma extending beyond the anterior canal wall and down to the tympanic cavity, which was filled with granulation tissue that was curetted. The malleus and incus were removed and the entire cavity cleaned. An area of necrosis was found superior to and anterior to the horizontal semicircular canal. This extended inward to a depth of one-third inch and forward above the semicircular canal and around it for a depth of one-half inch. It was removed. An opening was made one-sixteenth inch posterior to the mouth of the

eustachian tube, with a burr directed at an angle of twenty-two degrees to the external auditory canal inward and forward (as described in Section III of this paper). After trephining for a depth of one-half inch, a cavity was entered from which about one-half a dram of pus was evacuated, with debris and granulations. This cavity was cleaned out. Its depth measured from the posterior meatal wall to its termination, was approximately nine-sixteenth inches. The wound was packed with iodoform gauze and left open.

The patient was very restless after the operation and gritted her teeth continuously. The pulse was thready, but the pain in the eyes was relieved, only to return after, later in the day, accompanied by some swelling. There was a rise in temperature, from 101.8 to 105.2, and she was drowsy. The finger nails were cyanosed and the face pale. The quality of the pulse improved; but she complained of severe pains in the back and the right leg. She voided in bed. The next day, April 29th, the highest temperature was 104.4, from which it dropped to 99.2. She still complained of pain in the right eye, of severe abdominal cramps and of nausea, vomiting several times during the day, but she appeared less drowsy and more playful . . . the nystagmus was less pronounced, disappearing at times. The fundi were practically unchanged, except for some signs of recession in the right disc. The abdominal reflexes were equal. The right knee jerk was diminished and the left absent. Ankle jerks were active. There was the suggestion of a bilateral Kernig, but no rigidity of the neck. On April 30th the temperature range was low, between 99 and 99.8. A round, red area appeared on the right cheek, but she appeared brighter during the day. In the evening she complained of severe pain in the wound and abdomen and of headache. Both knee jerks were present. The Kernig on the right side was almost gone, although there was still a suggestion of one on the left. The left plantar response was equivocal. The right pupil was smaller than the left; and there was a narrowing of the right palpebral fissure. Ophthalmologic examination showed, in addition a blurring of the nasal discs with congestion of the blood vessels, but no evidence of hemorrhage or exudate.

The next day there was a rise in temperature to 101.6. She still complained of pain in both eyes and occasionally in the abdomen; but she was much brighter and more cheerful and appeared improved. On May 2nd she was very restless and irritable again, complaining of pain in both eyes and the neck, of severe pains and stiffness in the back and of occasional pain in the abdomen. The right eye was more frequently complained of than the left. The temperature stayed between 98.6 and 99.4 however, and she appeared much brighter.

During the next two days the temperature ranged between 99 and 99.8. The child continued to be restless and irritable and there were frequent complaints of pain in the right eye. The abdomen also was complained of occasionally. On May 5th there was a sharp rise in temperature to 104.2 and the child was again drowsy and nauseous. She complained of pain in the eyes, especially the right, and of occasional pain in the abdomen. Her neurologic status was unchanged, except that the nystagmus to the right was a little more pronounced. A spinal tap was done and 10 cc. of sanguineous fluid were removed, not under pressure. On the following day the temperature range was lower, between 99.4 and 101.8. The pain in the eyes was still present and that in the abdomen was more severe.

The red area which had previously been observed on the right cheek reappeared, and she complained of a general malaise. The abdominal pain and nausea was still complained of on May 7th as well as the pain in the eyes. The temperature stayed between 99.2 and 100.2. The next day the pain in the eyes persisted, worse this time in the left; but the abdominal pain was less. The temperature went no higher than 99.8 and the condition of the wounds was good. Her lips, which she had been biting, were swollen and painful.

On May 9th the principal complaint was of pain in the left eye and the left side of the mouth and lips; an ulcerative stomatitis was present. She did not complain of as much pain in the abdomen and appeared brighter. The temperature went to 101. The highest temperature on the following day was 100.2, but there was still a complaint of pain in the left eye. Eye examination revealed that the right pupil was still smaller than the left. The fundus picture was unchanged; the left eye was more pronounced than the right.

On May 11th the child was much brighter and more cheerful and the temperature did not go above 100.2. There was a red and sore looking area at the back of the head; but, the condition of the mouth and lips was improved. There was still pain in the eye. For the next ten days the condition of the patient slowly continued to improve. The temperature stayed down and the blood picture was better. The pain in the eyes gradually lessened in frequency, the left was more often complained of than the right. On one day the hip bothered her; on another a red area appeared on the right labia majora. The neurologic picture cleared, however, and there was a definite general improvement. Progress from this point was rapid, and on June 2nd the patient was discharged from the hospital. Convalescence continued uneventful. X-ray examination of the petrous bone taken one year postoperatively shows a productive osteitis of the petrous apex."

An analysis of this case reveals that swelling of the eye recurred repeatedly, followed by more serious symptoms. On April 7th, at the first examination, she was complaining of pain over the right eye, which was swollen. After the mastoidectomy that day the case ran a mild course, with the exception of continued ear pain and occasional retro-orbital pains. On April 17, the patient began to complain again of severe pain in the right eye, which appeared more swollen. From that time on we again have a stormy symptom, leading to re-operation and the removal of a thrombus from the lateral sinus. This procedure, however, did not influence the eye pains, which recurred. On April 24th, we again find an exacerbation of symptoms associated with pain of the right eye and some swelling of the eyes. This, however, the author attributes to crying. Although swelling of the eye had appeared twice before, yet very little significance is attached to

it. These signs were indicative of venous embarrassment of the anterior group, draining from the tympanic cavity to the pterygoid. On April 25th, in addition to pain in the right, both eyes were swollen. The following day the right eye and cheek were swollen. Following this there was a rise of temperature to 104.8 and severe symptoms until the petrous bone suppuration was evacuated and healed. The middle ear at operation was found filled with granulation tissue.

Case six of the same report of Kopetzky and Almour again illustrates the anterior venous congestion, associated with petrous bone involvement.

On analysis of this case, we observe that although a bilateral mastoidectomy was performed, the first symptom pointing to further involvement of the right side was swelling of the right eye. Twelve days later, after discharge from the hospital, there was a recurrence of symptoms on the right side which went on until relieved by operation and evacuation of pus from the petrous bone. At this point I wish to report the fourth case of my own series.

C. W., a girl of 7, referred to me by Dr. Brager, was first seen by me on February 2, 1931. She had developed an acute left otitis media, nine days previous. The drum membrane had been incised four days later. During the subsequent four days prior to my seeing her she complained of severe pains in the temporomaxillary region and cheek which had been variously diagnosed as neuralgia. Examination at that time showed a prominently bulged drum and profuse purulent drainage from the middle ear. There was at that time also slight puffiness of the left lower lid and pain in the left eye. She had an old strabismus which was not influenced by the present illness. On February 4, 1931, she was hospitalized and kept under observation for three days, the temperature ranged between 99 and 102. On February 7, 1931, a simple mastoidectomy was performed. At operation extensive pneumatization was found. The cells were coalesced so that the sinus plate was smooth, necessitating very little curetting. The zygomatic cells reached well towards the zygomatic root. These were carefully curetted and the mastoid cavity cleaned to the inner plate towards sinus, dura and zygoma. The wound was packed with iodoform gauze and closed with Michel clips. The subsequent two weeks were entirely uneventful with the exception of the complaint of occasional frontal headache on the left side. On February 18, 1931, she was discharged from the hospital and came to the office for dressings. Her middle ear had been dry from fifth day after the operation. On the second day after discharge from the hospital she began to complain of pain in the left eye coming on chiefly at night. Her appetite which had been excellent at the hospital became very poor. Her temperature ranged between 99 and 101. During the next three days the eye pains became

progressively worse. The attacks would come on spasmodically, chiefly at night about every half hour. These were so intense that on one occasion she tore her bed sheets. She vomited every morning and refused her food. On February 26, 1931, she was hospitalized again. That evening her temperature rose to 104. At twelve that night I observed puffiness of her lower lid. This indication led me to perform a wide paracentesis of the drum membrane in an effort to induce free bleeding from the middle ear according to the rational which I have conceived and described in this thesis. The middle ear had been dry and the drum membrane gray and apparently thickened. Following the paracentesis there was an unusual flow of blood but no pus. The next morning the ear was again dry. During the night the temperature receded, the eye pains improved. The next day the patient was much brighter, her appetite improved, the puffiness on the lid disappeared and during the succeeding days the patient progressed to recovery. At the onset of the eye pains the mastoid wound was opened partly to allow inspection, but no evidence of retained suppuration was observed. The opening of the flaps, however, necessitated a subsequent resuturing of the wound edges.

At first glance one would be disposed to question the significance of the edema of the lower lid, but in all tour of my cases it was the indication for early intervention on the middle ear. It is also quite obvious that in practically all of the cases of petrous suppuration the middle ear was found filled with granulations. The early relief of middle ear congestion is evidently an important factor in arresting involvement of the petrous bone. An additional factor pointing in that direction is that the symptoms of petrous bone involvement usually develop about a week or ten days after the mastoid operation when the drum membrane has closed and the middle ear is filled with dense congested granulations. In line with my conceptions regarding the importance of venous congestion of the middle ear after mastoidectomy and their causation of symptoms referable to the petrous tip, is a case reported from the clinic of H. Neumann in Vienna, at the meeting of the Austrian Otologic Society, January, 1928.

I wish to report three further cases indicating the sensitiveness of the nerve structures, particularly the Gasserian ganglion and abducent nerve, to vascular changes. This is illustrated in the fifth and sixth cases of my report.

A man, A. B., was seen by me on February 11, 1932. He was 33 years of age. He had a right acute otitis which had ruptured spontaneously about four days before. At the time of examination he was complaining of pain and fullness in the left ear. Examination revealed a marked bulging of the drum membrane with hemorrhagic bullae on the drum membrane and canal wall. Although the patient himself had not previously

observed it there was a slight paresis of the left abducent with definite diplopia on looking to the left. Incision of the drum was followed by profuse bleeding and drainage of sero sanguineous discharge. Two days later there was no evidence of the diplopia or abducent paresis.

Here we see a case of transient abducent involvement in the early stages of vascular engorgement of the middle ear. Had I not been especially interested in these phenomena, I would probably not have observed this.

The next case, however, is not an otologic one, but is inserted to indicate the early reaction of the Gasserian ganglion to venous engorgment, when associated with embarrassment of the pterygoid circulation. On February 23, 1932, I was called in consultation to see Miss F., age 21. On Saturday she felt what seemed to be a small pimple on the inside of her right upper lid. On Sunday she noticed some swelling of the lid and had some headache. On Monday the eye became more swollen, in the morning she had vomited once. During the day she complained of pain in the upper and lower teeth as well as the eye region. This pain in the teeth she ascribed to neuralgia. She also had some difficulty in opening her mouth. Apparently due to trismus of the muscles of the right side. When seen on Wednesday she had proptosis and chemosis of the conjunctiva. Nasal examination revealed no evidence of suppuration. X-ray showed slight clouding of the right ethmoid and sphenoid but no exudate. This diminished illumination was probably due to the swelling of the eye. There was still some complaint of pain in the teeth and definite trismus. An orbital phlegmen was diagnosed, but in addition, I pointed out that there was stasis in the pterygoid plexus, giving the trismus and venous engorgement of the Gasserian ganglion and that we would probably be getting cavernous signs shortly. Examination that evening by Dr. Arnold Knapp showed a small amount of necrotic tissue on the nasal side of the orbit and an orbital phlegmon was diagnosed. The next day definite signs of cavernous sinus thrombosis developed.

The practical application of these conceptions is demonstrated in this concluding case of my report.

Rabbi F. N., a patient of Dr. Harold Hays, whom I saw in consultation April 11, 1932, following an attack of grippe which lasted about two weeks, he experienced noises and discomfort in the right ear. Shortly thereafter the left ear began to discharge spontaneously and he developed a fever ranging between 100 and 101. This was associated with pain in the left side of the neck and generalized headache. The left ear discharge stopped but the fever continued. On March 23, 1932, a paracentesis was performed and on the 28th a left mastoidectomy was carried out. A sclerotic cortex was found, the mastoid containing very few cells. The antrum and cells down to the tip were found filled with pus and were cleared out. A slight nick of the jugular was controlled by packing. The patient improved and was discharged from the hospital on April 6th. During the time at the hospital the temperature range was between 100 and 100.5. The culture of the pus showed streptococcus hemolyticus and staphylococcus aureus. One of the blood counts showed 2 per cent myelocytes, but did not recur in subsequent examinations.

On the day of the mastoidectomy on the left ear he noticed a shift of the pains to the right ear. This continued to annoy him intermittently, radiating from the temple to the occiput. The pains were more in the head than in the ear. When he had his pains in the temple it radiated to the face and jaw and for two days was severe enough to cause him difficulty in eating because he couldn't open his mouth on the right side and had to slip the food into his mouth.

At the time of leaving the hospital, although the left ear did not bother him, he had a thumping sensation in the right supraorbital region. After the discharge from the hospital he felt fairly well for about two days when he suddenly developed a temperature of 102. The next day he had a chill while coming to the hospital. That evening he had pains in the back of the head and the temperature rose to 104, pulse 124, respiration 26. At six o'clock that evening the temperature was 102.

The next day, April 11, 1932, when I saw him in consultation, he was complaining of severe right orbital and supraorbital pains radiating to the right temporomaxillary region. He was restless and slightly nauseated. Examination showed a slight nystagmus to the right, although the day before it had appeared to the left. The general appearance of the man was that of an acutely sick patient. The right middle ear showed no evidence of acute infection. The drum details were visible, the malleus appeared retracted but in the posterior quadrant was a slight yellowish discoloration. Considering the earlier history of temporomaxillary pain and trismus, although no edema of the low lid had been observed, and the present findings of the severe orbital and supraorbital pains in conjunction with the suggestion of old pus in the middle ear, I diagnosed venous involvement of the petrous bone and advised free bleeding of the middle ear by paracentesis. Incision of the drum membrane was followed by profuse bleeding. The next day was marked by a definite purulent discharge. The relief from the severe orbital pains came shortly after the paracentesis and the next day the patient was quite comfortable and the fever was lower. The following day, however, came a recurrence of the orbital pain and rise in temperature. On April 14th Dr. Hays performed a mastoidectomy on the right side. At operation but few cells were found in the mastoid, those near the sinus showed some pathology. Following mastoidectomy, however, the orbital pains continued without much relief. The patient continued running a course of intermittent orbital pains and a temperature fluctuating between 100.5 to 101.5. This course continued for several days when he began to show slurring of speech, coated tongue and slight signs of cerebellar ataxia and nystagmus to the right. A cerebellar abscess was diagnosed. The mastoid wound was again exposed and a radical mastoidectomy performed. The middle ear was found filled with granulations. The tegmen tympani was somewhat necrotic and the dura covered with granulations. The dura of the posterior fossa was uncovered but no pathology found. Because of the progression of the cerebellar symptoms the cerebellum was exposed by the posterior route and a right cerebellar abscess located. The apparent absence of a cerebellar pathway of infection from the mastoid and the presence of the signs of petrosal involvement suggest extension to the cerebellum from the petrosa possibly below and medial to the external auditory meatus.

In this case, although the patient is still under care at the time of this writing, nevertheless, the relief of the orbital and temporal pains, as well as the nausea and slight nystagmus, the return of his appetite and improvement in well being, after the first paracentesis and bleeding, was almost dramatic in its appearance. It demonstrates the direct relationship of the symptomatology of the syndrome which I am describing, and the proof of the accuracy of the conception in the therapeutic test.

Let us now look closer to the mechanism of the pain in petrosal involvement. One of the most outstanding symptoms is the orbital pain. In the posterior group presenting the Gradenigo syndrome of abducent paralysis there may be orbital pain preceding or associated with the palsy. In the anterior type which I am describing the orbital pain usually follows a preceding period of temporomaxillary pain and trismus. The understanding of these clinical symptoms again lends itself to exact anatomic explanation. The ophthalmic nerve passes forward to the orbit through the middle fossa of the skull beneath the dura mater. It lies in the lateral wall of the cavernous sinus, at a lower level than the trochlear nerve and reaches the orbit through the superior orbital fissure. In the wall of the cavernous sinus the ophthalmic nerve gives off (1) a small recurrent branch to the dura mater (n. tentorii), (2) communicating branches to the cavernous plexus of the sympathetic on the internal carotid artery, and (3) small communicating twigs to the trunks of the oculomotor, trochlear and abducent nerves. In the superior orbital fissure the nerve divides into three main branches—lacrimal, frontal and nasociliary (Cunningham). We see here a rather direct connection of the ophthalmic nerve with the nerve mechanisms of the petrous bone, both in its supply to the abducent nerve itself as well as to the sympathetic mechanism around in the carotid canal and the dura. Both the venous congestion of the middle meningeal venous drainage to the pterygoid and the venous mechanism of the inferior petrosal in its relation to the abducent could, therefore, give rise to the typical ophthalmic pain in the eye and frontal regions. The relief of these symptoms resulting from decongestive procedures in the tympanic cavity added further support to this anatomic explanation. It is remarkable that the mastoid operation itself will

usually not afford as much relief from the eye symptoms as the intervention on the middle ear, thus again correlating petrosal symptoms to the tympanic cavity.

I have pointed out that in the anterior type of involvement we usually get temporomaxillary pains and trismus, which is transient and associated with edema of the lower lid. These symptoms may be of very short duration and are replaced by the more prominent orbital pains of petrosal involvement. This mechanism also has its anatomic basis through the congestive state of the middle meningeal veins, influencing the mandibular nerve rather than the ophthalmic. The mandibular nerve as it emerges from the skull through the foramen ovale to the pterygomaxillary fossa gives off a small nervus spinosus which arises just outside the skull and accompanying the middle meningeal artery through the foramen spinosum supplies the dura mater. In the infratemporal region a small branch arises for the supply of the internal pterygoid muscle. This nerve forms a connection with the otic ganglion. The buccinator, which is also sensory, passes forward between the two heads of the external pterygoid, is distributed to the skin of the cheek, giving the face pains which at times are observed for a short while.

A still further supply exists through the auriculotemporal branch of the mandibular nerve, which gives off (1) a small branch to the mandibular articulation, (2) a twig to supply the skin of the external acoustic meatus and membrana tympani (n. meatus auditorii et ramus membrane tympanii), (3) branches to the superior half of the auricle on its lateral aspect, and (5) terminal branches to the skin of the temple and scalp.

We see from these relationships of the mandibular nerve to the middle ear that symptoms referable to the members of the Gasserian ganglion need not represent actual pathology at the petrous apex, but may be indicative of a congestive state in the middle ear which both creates symptoms referable to the Gasserian ganglion and vascular embarrassment of the petrous bone leading in some cases to extension and suppuration.

It also shows how apparently severe symptoms, such as demonstrated in Schlander's and Neumann's cases, leading them to suspect grave involvement of the petrous apex, improved all too

remarkably by the simple procedure of paracentesis and calls for the greatest caution in instituting radical intervention on the petrous bone.

CONCLUSIONS.

In concluding, I wish to point out, first, that the venous pathways of the temporal bone play a leading rôle in the dissemination of infection from the tympanic cavity and the causation of intracranial complications; second, that early extension of involvement of the venous system can be recognized clinically and should serve as a guide for early accurate intervention; third, that the Gradenigo syndrome should be considered as symptoms of venous engorgement of the group of tympanic veins emptying into the inferior petrosal sinus; fourth, that the syndrome of temporomaxillary orbital pain, trismus and edema of the lower lid, which I have described, is of similar significance with the Gradenigo syndrome, but represents venous engorgement of the veins of the tympanic cavity draining anteriorly into the pterygoid plexus and middle meningeal vein. Clinically, I have shown that the early incision of the drum membrane and the induction of free bleeding from the middle ear afford relief from the symptoms and may obviate suppuration of the petrous pyramid. It must not, however, be construed that paracentesis and free bleeding from the middle ear will relieve advanced involvement of the petrosa with suppuration and bone coalescence.

Finally, one must bear strictly in mind that the orbital and trigeminal symptoms may be induced by mechanisms affecting the pterygoid and middle meningeal venous systems other than of petrosal origin, and must be carefully differentiated to avoid unwarranted surgical invasion of the petrous bone.

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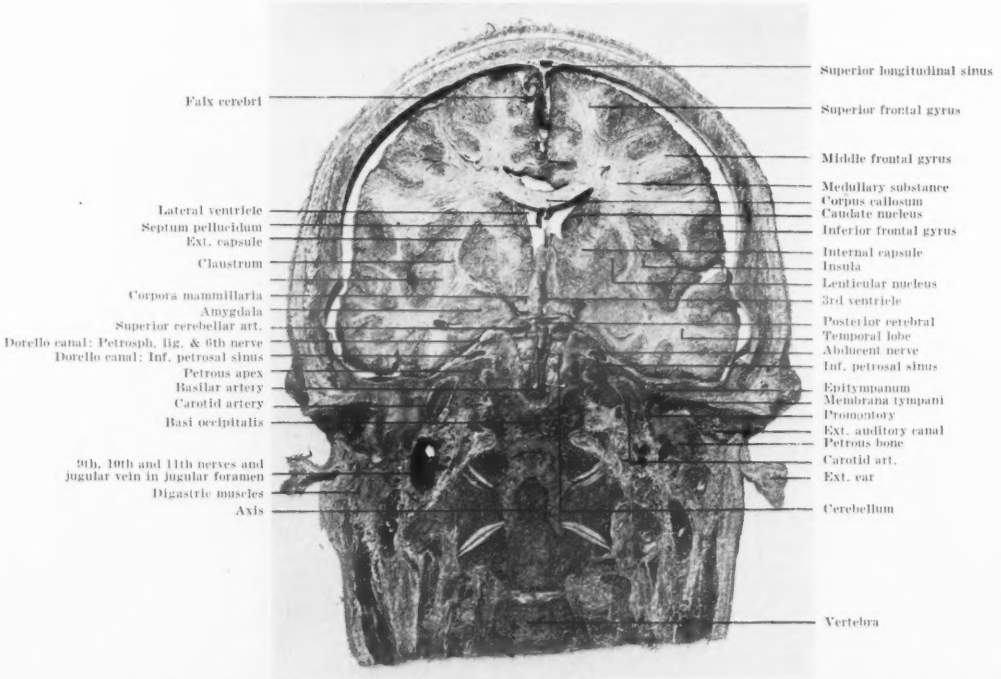

Fig. 1. This demonstrates clearly the Dorello canal, showing the petrosphenoid ligament running from the apex of the petrous bone to the sphenoid having in the angle between the ligament and the petrous bone, the inferior petrosal sinus which one sees very clearly on the left side and above on the abducent nerve which has not been labeled on the left side in order to avoid obliterating marks, but, it is revealed on the right side. One can readily see how the engorgement of the inferior petrosal sinus would directly encroach upon the abducent nerve.

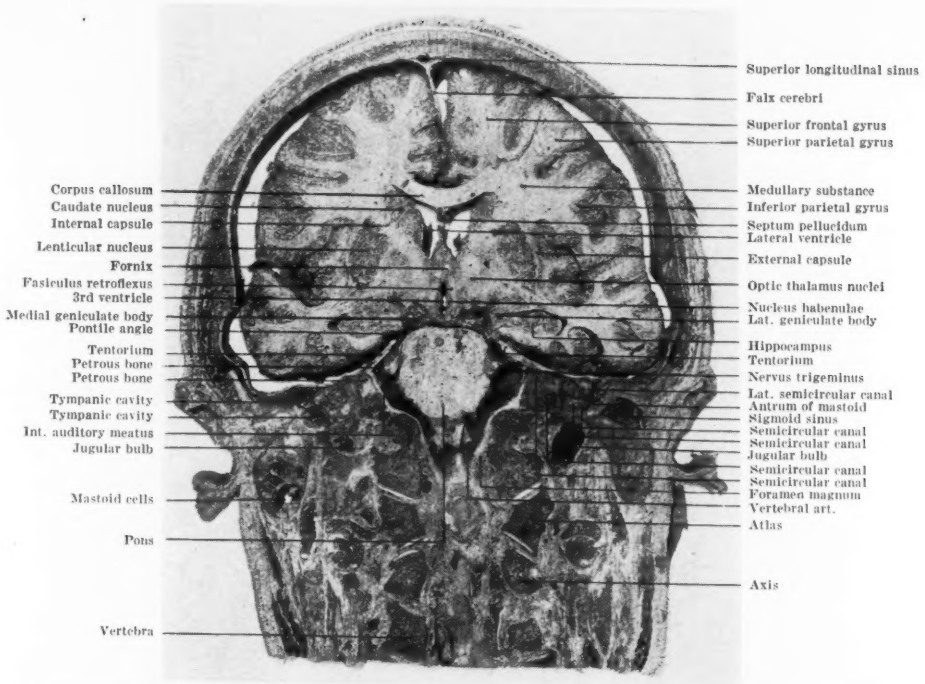

Fig. 2. Demonstrates the cellularization of the petrous bone and its relationship to the labyrinth. It shows clearly the intimate connection between the tympanic cavity and the pneumatic petrous cells.

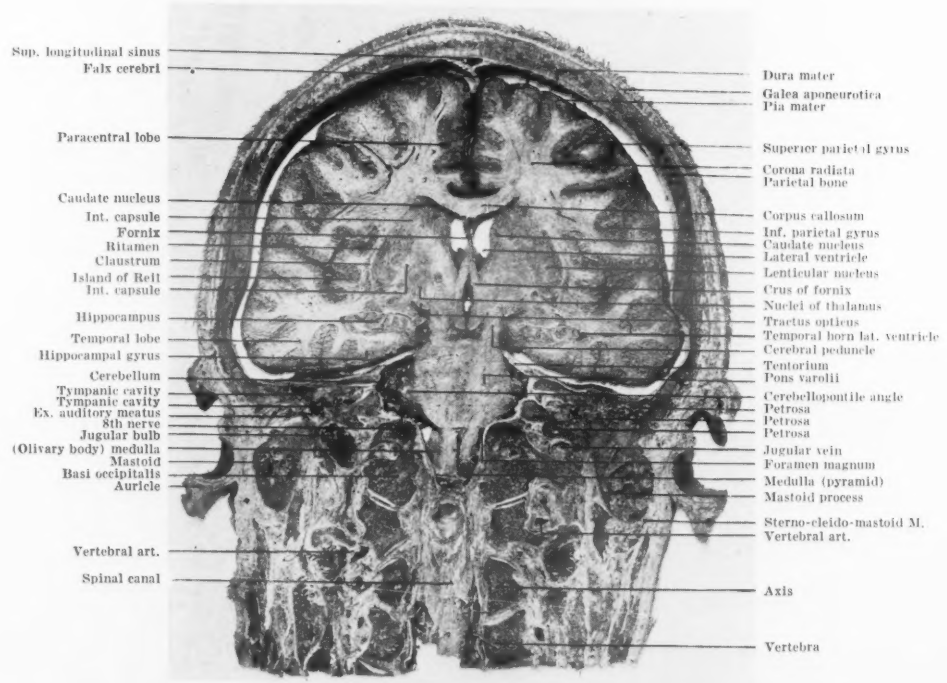

Fig. 3. Shows the cerebello pontile angle and its relationship to the pneumatic cells of the petrous bone. This is of especially great significance.

Fig. 4. Shows the relationship of the labyrinth to the cellularization of both the mastoid and petrous areas.

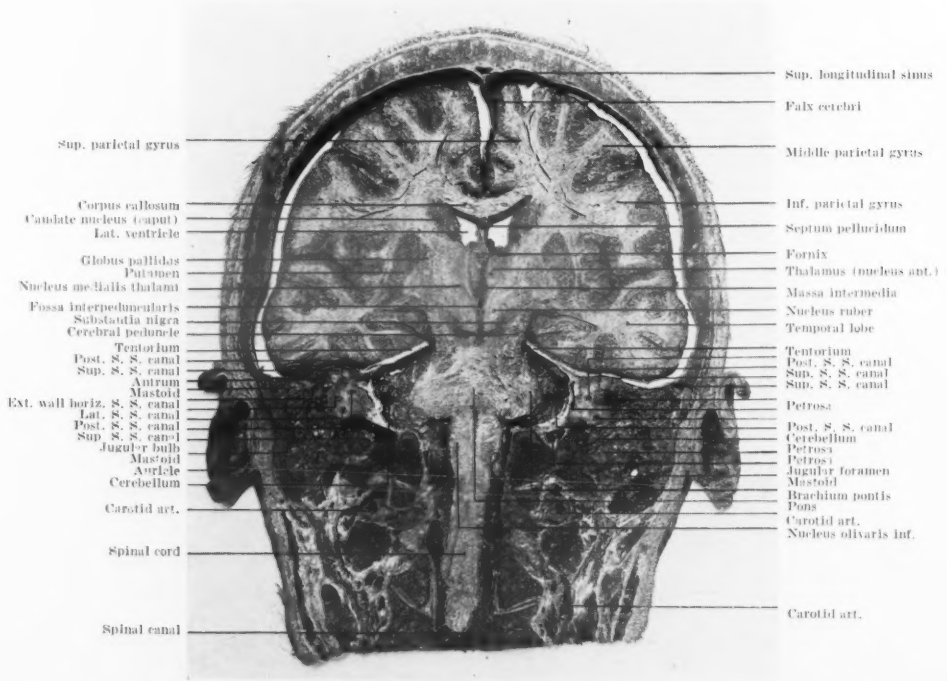

Fig. 5. Demonstrates remarkably the relationship of the carotid canal and labyrinth to the cerebello Pontile angle.

LXXIX.

A DISCUSSION OF CARDIAC, PULMONARY AND OTHER GENERAL CONDITIONS SECONDARY TO CHRONIC NASAL SINUS INFECTION.*

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OMAHA, NEB.

The serious general effects of acute and chronic infection of the nasal accessory sinuses are not appreciated by the laity and by but a very small part of our profession. In fact, in recent years there has been a wide tendency by the general profession to disparage the rhinologist and his work. Undoubtedly some of the criticism is justified, and we are partially to blame for much of that criticism. The fundamental basis of our trouble is that no regulations have been made providing for the qualifications and special preparations necessary before beginning the practice of a specialty, and until we are equipped as a specialty to render the expert service required of us, we will not command the confidence of the profession and public. At present too many diagnoses are missed and too much treatment is misdirected or inadequate, consequently not giving relief, or the surgery is often meddlesome and should not have been done. Therefore we have not rendered the profession the service that is expected and hence it follows that many lives have been sacrificed, and misery and suffering that could have been relieved has and does exist. As an illustration of the feeling that does exist, I call to your attention an article by Dr. C. M. Anderson, an internist in one of our great clinics ("Suppuration in Paranasal Sinuses as a Factor in Focal Infections: Review of 400 Cases," J. A. M. A., 1930, XCIV, 1889-1891), in which he seems to conclude that infection of the nasal accessory sinuses plays a very small rôle as a causative factor in focal infections. This view is all too common, and while

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many others recognize the importance of infection, they are doubtful of our ability to relieve it.

For a number of years I have been working in close affiliation with the Department of Clinical Research of the University of Nebraska College of Medicine. Our work has led us to the conclusion that infection of the nasal accessory sinuses plays a very important part in general systemic diseases. Indeed, there is scarcely an organ in the body that we have not found affected.

The general appearance of patients often marks them. They look worn and tired; they have lost their snap, and life has lost its zest. Their eyes look tired, the lids puffy; the skin has lost its healthy glow and they are mentally fagged; their shoulders are drooped and they walk flatfooted; they are easily irritated and find it difficult to concentrate. The diagnosis from a rhinologic standpoint presents many difficulties. I think it is quite important to have a general working classification, and for my own use I have the following:

1. Acute.
2. Chronic purulent with frank pus in the sinuses but little thickening of the mucous membrane.
3. Chronic hyperplastic, with or without polypi, and a very sticky secretion, often only scanty in amount.
4. Mucocele.

I will not discuss types one and four. In type two, the turbinate bones adjacent to the infected sinuses are usually tumefied and more or less block the nares. Purulent secretion can usually be seen. Radiographs show the affected sinuses diffusely dense and frequently a fluid level, but usually not much thickening of the mucous membrane lining the sinuses. Type three shows a wide variety of pathology of the nasal structures. The turbinates range from no visible pathology to polypoid degeneration. The color of the mucous membrane varies from pink to almost white, seldom red. Polypi may or may not be present.

Proetz's method of displacement with opaque oil is a most important aid in diagnosis, as it will usually show the presence of hyperplastic membrane (Tr. Am. Laryng. Assoc., 1930, LII, 121-140). The maxillary sinuses, in our experience, are most sat-

Fig. 1. Case No. 32. Lipiodol injection. Waters position. Note thickened membrane and discrete abscess in membrane of lateral wall of left antrum.

Fig. 2. Case No. 101. Lipiodol injection of maxillary sinuses. Note thickening and polypoid changes in the membrane.

isfactorily filled by catheterization of the osteum. Diagnosis of the hyperplastic type is most essential, as it is the one that generally requires a radical operation. The other classes often respond quite satisfactorily to treatment or intranasal operation.

It has further been our experience that hyperplastic sinusitis is frequently made worse by intranasal surgery. This is especially true of the maxillary sinuses. We have also found that a large opening below the inferior turbinate does not allow the membrane of the sinuses to return to normal. We have repeatedly found this sinus with a free opening which has been present for months or years, and still the membrane contains multiple abscesses with both grossly and microscopically the same evidence of infection as though no such opening existed. In many such sinuses it is possible to see these abscesses before operation by means of a pharyngoscope. It is this hyperplastic type that is most frequently associated with systemic diseases, especially with asthma.

As pointed out by Proetz and others, there is a chance for error in diagnosis when opaque oil is used in the sinuses due to an acute edema, allergic swelling of the mucous membrane, acute exacerbation or any acute inflammation. This must be kept in mind, and if there is any doubt as to the diagnosis, the true condition can usually be established by further investigation of the history, radiographs, tests for allergy and time.

Grossly these membranes are thickened, usually ranging from 20 to 40 millimeters in thickness. Polypi are frequently present, sometimes large and almost filling the cavity and at other times they are small, resembling a carpet of softly rounded masses. The deeper portions of the membrane are tough. Very frequently discrete abscesses, from pinhead size to that of a bean, are scattered throughout the membrane. These are quite as liable to be found on the upper outer wall as in the floor. Occasionally one is seen that contains thirty or more minims of pus. Intranasal drainage of such a sinus will not reach these abscesses. The secretions or fluid present in these sinuses is usually very thick and sticky, almost like raw rubber. A diagnostic puncture with irrigations frequently will not remove this secretion.

Fig. 3. Case No. 32. En masse exenteration of both maxillary sinus membranes. Coronal section. The material has been removed in one piece and the membrane is complete except for the usual tearing along the medial wall near the ostium. Note the abscess in the left antrum (Fig. 1).

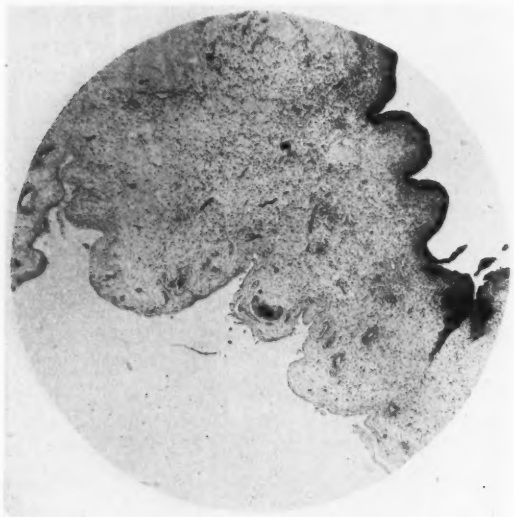

Fig. 4. Case No. 101. Low power of portion of maxillary sinus membrane showing fibrosis of submucosal layers and thickened blood vessels in lower left portion while in upper portion there is acute swelling.

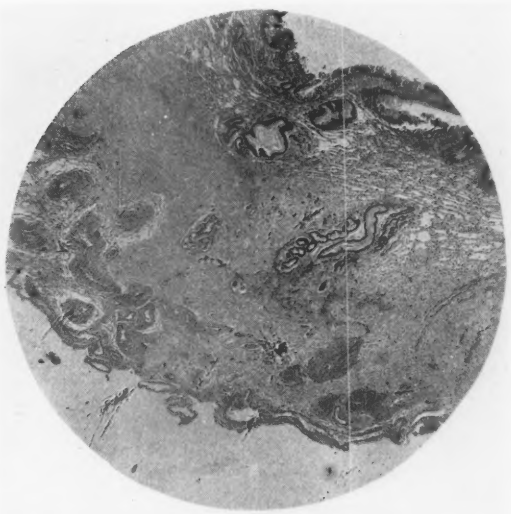

Fig. 5. Case No. 8. Low power of portion of maxillary sinus membrane illustrating the variety of pathological processes which may be present in a single field.

Microscopically the membrane shows a tough base, rather dense and containing many connective tissue cells, fairly numerous blood vessels, and as we go to the surface the cells are less numerous and there is a greater amount of fluid space. But this is not constant and areas of fresh proliferation are not through the whole membrane. The more chronic the condition, usually the more hyperplasia of cells. In acute swellings, the tissue is very edematous and tender. (The bone in old chronic hyperplasia is usually softened.)

Most important of all is the presence of bacteria all through the membrane. These can be shown in section by special staining methods. This, then, is the kernel of the whole problem. The infection as shown by the abscesses and bacteria in the membrane itself explains why our former efforts at drainage and incomplete removal were unsuccessful in relieving metastatic infection. They also make plain the necessity of complete removal of the infected membrane. This is most effectively done in the max-

Fig. 6. Case No. 5. Higher power showing the hydrops and epithelial proliferation together with the inflammatory infiltration.

illary sinuses and hence to date our results have been best when the infection was confined to this sinus.

Fortunately, the membrane on the septum and the turbinate bones seems to escape this degeneration or is able to return to normal after the sinuses are relieved of their infection. The frontals lend themselves to operation almost equally as well, but the ethmoid and sphenoid are more difficult. Still they can be very effectively dealt with, due to their better natural advantage of drainage, and in most cases give very satisfactory results. As a source of infection the secretion found in this type of sinus is, I think, relatively unimportant, as the infection is a true bacteremia from the infection in the sinus membrane, with the toxins and bacteria entering the system by the blood stream and lymph channels directly from the membrane and not absorbed from its surface. Hence, again the futility of irrigation or drainage.

I use local anesthetics for all my operations, a nerve block preceded by morphin and scopolamin. In five years of this practice I

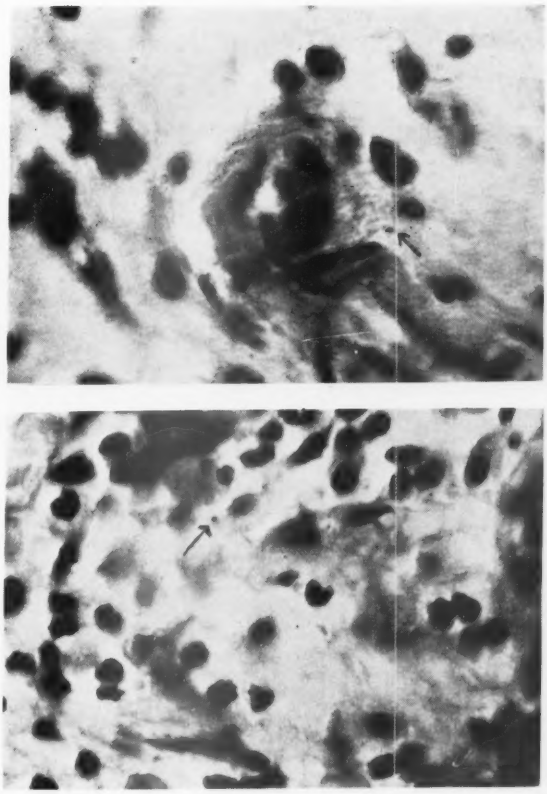

Fig. 7. Diplococci indicated by arrows. Gram-Weigert stain.

have had no serious complications. A few patients have had an excitement state lasting from two to eight hours following the operation.

For the radical type of operation on the frontals, ethmoid and sphenoid, I have found the one advocated by Lynch the most satisfactory. For the maxillary sinus, the Caldwell-Luc with some modification. The incision should not extend anteriorly beyond the second bicuspid tooth and should be made sufficiently low so that the periosteum can be elevated without exposing the fat in

the cheek. This prevents discoloration and swelling of the cheek and lessens the chance of infection of the soft tissues to a minimum. In removing the membrane of the maxillary sinus, I lay particular stress upon removing it en masse by very careful dissection with blunt elevators, avoiding entirely the use of curettes. This seems to be important, as sections made of the bone so denuded show a very thin layer of vascular tissue left over the bone. This, I feel, assists in the healing and may, at least in part, be responsible for my not having had to date any cases of osteomyelitis. Gentleness in the dissection and handling of all the tissues is one of the most important factors in the success of the operation. The operation is greatly facilitated by having a suction apparatus which can be regulated to about three to four pounds vacuum and a small suction tip. (I use Walker's submucus elevator and dissector.) This is kept back of the mucous membrane and keeps the field clean. If forcible suction is used, it is liable to damage the specimen and spoil the dissection. No sponging is used in the sinus during the operation or afterwards, on antiseptics and no packing. The ethmoid cells around the osteum should be carefully examined and any that are degenerated removed.

Fig. 8. Case No. 93. Maxillary membrane removed with portion of bone attached, and showing a natural plane of cleavage in the nasal layers in which a thin layer of tissue remains attached to the bone.

Postoperative care, ice over the sinus operated on for one day and following this all the heat possible for seven to ten days. No nasal treatment except albolene, sprayed or dropped into the nares frequently. No irrigation except occasionally one or two to wash out a clot from the maxillary sinus on the fifth to the

seventh day. Operate on only those cases that have undergone extensive chronic degenerative changes. Strive for accurate, clean, gentle, en masse enucleation of the sinus membrane.

The following case reports illustrate some of the different conditions, apparently secondary to chronic accessory nasal sinus infection:

Case 1.—Mr. C. W. H., aged 65, consulted me April 15, 1930. Had been home for a few days with a bad cold but now back at his business. Feels tired and irritable, can not concentrate, eyes puffy, has no headaches. General examination negative. Purulent left maxillary sinusitis. Recovered rapidly under local treatment.

Case 2.—Mr. M. A. H., aged 69, came in December 1, 1930, with the history that for the past two years he had suffered with nasal obstruction. Has had previous intranasal operations without relief. One week ago had a sudden attack of aphasia, in bed six weeks. Physical condition remained much below par, unable to attend to business and mentally disturbed. Now has definite cardiac decompensation and myocardia. Radiograph with opaque oil, maxillary sinuses, marked thickening of the membrane, ethmoids dense, frontal sinuses apparently not involved. Exenteration of antral mucous membrane and ethmoid cells around the osteum on December 3, 1930. This was followed by a fairly rapid recovery. He was able to resume his practice of law in two months and has remained well and moderately active to the present time.

Case 3.—Mr. C. W., aged 57, seen in consultation on September 21, 1932. He was very much depressed with delusions of self-unworthiness and paranoidal ideas that someone was going to harm him. During his stay in the hospital, showed marked mental confusion that suggested a serious organic type of dementia. Radiograph, chronic hyperplasic sinusitis. Following a Caldwell-Luc operation on September 26, his mental condition cleared fairly rapidly. He left the hospital October 27 with his mental condition relieved, and has remained in good condition to the present time.

Case 4.—Mr. R. O. C., aged 46, has had asthma for several years. On entering the hospital his asthma was so severe that he was unable to lie down day or night. Condition desperate, cardiac involvement and very cyanotic. Treated in hospital for two weeks, during which time he received large doses of epinephrin from four to eight times daily, sometimes with morphin. Complete radical operation, bilateral, on January 23, 1933. Considerable discomfort for the first 24 hours. Since his operation he has had no more asthmatic spasms, has not required any epinephrin or morphin, and has gone back to his position as drug clerk.

Case 5.—Mrs. G. O. M., aged 22, October 23, 1931, has had asthma since twelve years of age. Had several operations for removal of polypi and intranasal drainage of antra. Now has both nares filled with polypi. Free opening into antra below inferior turbinates, antra filled with polypi. On November 3, 1931, radical operation of the right frontal, ethmoid,

sphenoid and maxillary sinuses. Some weeks later, the same on the left side. Since then has only had a slight return of an attack of asthma for a few nights when she had an acute cold. Nose now free from infection, no scabbing or secretion, no polypi.

Case 6.—Mrs. D. P., aged 23, August 3, 1929, has had very severe asthma for the past six months which completely incapacitated her. First attack of asthma in fall of 1926. Is subject to frequent colds. Turbinates all tumefied, some postnasal mucoid discharge. Radiograph, antra marked shading, other sinuses clear. Bilateral exenteration of antral mucous membrane. This was thick and very fibrous. The asthma cleared up rapidly. Our last report from her on January 30, 1932, stated that she had not had any bad attacks of asthma and was feeling better than she had for years.

Case 7.—Mrs. O. C. (widow), aged 46, July 1930, since attack of influenza in 1928 has never been well. Has been subject to some chronic nasal catarrh for several years. She was very thin, pale, listless and depressed. Had been hospitalized for diagnosis and treatment three times in the past two years. Complained of weakness, pain in abdomen, constipation, some headache, sensation of fullness and ringing in ears. Has been diagnosed as a neurasthenic. Examination of nose, no gross pathology, mucous membrane rather pale. Radiograph, both maxillary sinuses, mucous membrane very thick, ethmoids doubtful, frontals and sphenoids clear. Bilateral exenteration of antral mucous membrane, July 7, 1930. This was thick and tough, contained multiple small abscesses and one large abscess in the upper outer portion of left sinus. Four months later she had recovered her normal health and remained well until the midsummer of 1932. In the meantime she had gained forty pounds. During August, 1932, she contracted a very severe cold and following this had some of her old symptoms,—stopped up head, bronchial cough and mental lethargy. In March, 1933, she again reported for examination. She now had definite evidence of infection of the ethmoids. I did an intranasal ethmoidotomy on April 18, 1933, and following this she made a good recovery.

For the lack of time I can not report more cases in detail.

SUMMARY.

Our experience has been that bronchial conditions usually respond well.

Arthritis, neuritis and allied conditions, except multiple arthritis and arthritis deformans, usually respond favorably.

Cardiac response is good.

Facial neuralgia, chronic headache, tinnitus, vertigo and ocular infections often are completely relieved.

Patience and study in this field of medicine yield many satisfactory results to both the rhinologist and his patient.

CONCLUSIONS.

1. In chronic hyperplastic sinusitis the infection lies in the membrane itself.

2. The secretion in these sinuses is relatively unimportant for diagnosis or therapeutic effect on local and general systemic disease.

3. The complete en masse removal of the membrane is the rational surgical procedure.

4. Gentleness of manipulation is essential to avoid complications.

I wish to thank Drs. A. D. Dunn, Lowell Dunn, Frederick Niehaus, Miss Helen Strothers and Miss Mildred Braden for their collaboration.

MEDICAL ARTS BLDG.

LXXX.

FULMINATING LARYNGO-TRACHEO-BRONCHITIS.*

LYMAN RICHARDS, M. D.,

BOSTON.

During the past few years, a number of children exhibiting variations and stages in development of a general disease picture of acute fulminating laryngo-tracheo-bronchitis have presented themselves at the Children's Hospital in Boston.

The course of this disease is so severe and the treatment so limited in possibility and yet so definite in indication that only a correct understanding of the underlying pathologic process can give a true interpretation of the presenting signs and symptoms, and thus permit intelligent handling of the case, and an accurate prognostic statement. We have been able to formulate a definite disease entity from a clinical and pathologic study of eleven cases recently encountered.

The effective handling of cases of acute upper respiratory obstruction rests fundamentally on two all important points: first, a correct diagnosis of the cause of the obstruction, and second, application of the proper treatment in view of this cause. In certain urgent types there may be necessary a reversal of order in the procedure and measures for relief of the obstruction may be required before the etiologic factor is known. Emergency tracheotomy in this situation is universally familiar. In a majority of instances, however, there will usually be time to ascertain, at least to a sufficient degree, the nature of the obstruction, and if this is possible untold advantages accrue to the operator in the decision as to future measures of treatment. It is only by examination of the larynx by direct laryngoscopy that one determines most effectively what the situation is with which he has to deal and by which he must be guided in subsequent treatment.

The three outstanding conditions which produce the situation of persistent acute upper respiratory obstruction are (1) foreign

*Presented at the meeting of the American Bronchoscopic Society at Washington, D. C., May 10, 1933.

body, (2) diphtheria, and (3) acute infectious laryngitis. The characteristics of the first two are so well known and have been so thoroughly considered by numerous authors as to warrant no further consideration here. The condition of acute infectious laryngitis with its frequent extension into the trachea and bronchi has, however, certain important aspects about which relatively little has been written and which need further consideration.

THE DISEASE PICTURE.

Fulminating laryngo-tracheo-bronchitis may be defined as an acute infection of the upper respiratory tract, characterized by a high febrile reaction, and an intense inflammatory change in the mucous membrane and walls of the trachea, bronchi and bronchioles and accompanied by the formation of a sticky, gummy, often glue-like exudate or secretion, partially or completely occluding the upper airways. The occluding secretion may extend upward to become attached to the glottic walls or the infection may proceed downward to fill the terminal bronchioles and alveolæ and hence to produce a bronchopneumonia.

Symptomatically, the onset of the disease is quite typical and characteristic. A child, most often between the ages of 2 and 4, in previously good health except for possibly a slight head cold, wakes up at night with the symptoms of spasmodic croup. There is the usual inspiratory stridor with hoarseness and often the barking cough which make up this well known clinical picture. Foreign body in the larynx, always a possible diagnosis with this symptomatology of sudden onset, can usually be excluded when symptoms arise after the child has been put to bed in perfect comfort. Classical treatment for spasmodic croup, usually and naturally applied to the situation, does not relieve the obstruction, and, on a physician being summoned, the next natural inference is diphtheria, for which antitoxin is quite logically administered until cultures can substantiate this diagnosis. By this time, however, with the laryngeal obstruction becoming more distressing, as manifested by increasing dyspnea with substernal and suprasternal retraction, the physician wisely decides on transfer to a hospital, where the one and only procedure which can hope to establish a correct diagnosis is direct laryngoscopy. By this pro-

cedure, a foreign body can be ruled out or removed if present, diphtheria can be diagnosed and its membranes eradicated by suction or forceps, and the obstructive crusts attached to the cords or glottic wall in the disease we are discussing can be likewise removed. These crusts, composed of dried secretion, can be seen as soon as the larynx is exposed to direct view. There is no bleeding on removal, and they have none of the other characteristics of true diphtheritic membrane. The supraglottic tissues, arytenoids and false cords usually show relatively little reaction of an inflammatory nature in contradistinction to the picture of laryngeal edema. The dry crusts will usually be found loosely attached to the walls of the subglottic tissues, from which they can be removed mechanically. Below this point the tracheal secretions are usually fluid in character and tend at first to remain so till, coming in contact with the air in the region of the larynx, they become dried and hence transformed into obstructive crusts.

Once these crusts have been removed from the larynx, there is a restoration of a free airway, the patient's condition greatly improves, signs of urgent dyspnea disappear and one is tempted to think that no further difficulty will arise. If in addition an intubation tube is inserted by direct vision into the larynx, an open airway is still further assured. Certain milder types of this infection will require no further treatment beyond close observation in event that the intubation tube is coughed up, and the forced administration of fluids by hypodermoclysis, since with an intubation tube in place many children refuse absolutely to take fluids by mouth. It is impossible to stress too strongly the importance of maintaining a high fluid intake, since only by this means can one combat the further tendency in these patients for the tracheal secretions to become dried out and hence to produce further obstruction. No amount of the usual expectorant medications will take the place of this forced fluid administration, since expectorants will fail unless the body fluids are present for excretion into the trachea.

A certain group of these patients, however, will prove to be only temporarily relieved by intubation. Comfortable for a few hours only, all the original signs of respiratory obstruction return with the agonizing restlessness, thrashing about and tendency

toward cyanosis. The temperature rises rapidly, the pulse rate advances to an average of 150 and the respiratory rate is close to 55 or 60. We have now the typical picture of a full-blown case of fulminating tracheobronchitis.

On first consideration one might feel that the site of this recurrence of obstruction again lies in the larynx and that the intubation tube, having become occluded, is no longer providing an adequate airway. Such, however, has not proved to be the case, since on removal the tube will be found patent with an open lumen. Likewise, if performed at the outset instead of intubation or if done on discovery that intubation is inadequate, tracheotomy will meet with little greater success, since either procedure accomplishes only the same thing—namely, relief of laryngeal obstruction. The fundamental fact is this—that the obstruction, due to accumulated dried and crusted secretions and to the descending nature of the inflammatory process, is now lower down, in the region of the lower trachea, tracheal bifurcation and primary bronchi, at which point no means designed to relieve laryngeal obstruction will be of the slightest avail. One procedure, and only one, can be utilized in this urgent situation, and that is bronchoscopy, with mechanical removal of the obstructing masses by suction or by forceps. Failing this, the bronchial lumen on one or both sides will rapidly become obliterated, air will be unable to reach the periphery of the lungs, steady asphyxiation will ensue in spite of everything that can be done, and the patient will die a death of suffocation, than which there is no more ghastly clinical spectacle.

Tracheotomy has, however, a secondary value in the event that matters reach the degree just described. Since a single bronchoscopy will rarely be sufficient in treating these cases, owing to the tendency for the crusts to reform and reaccumulate, repeated bronchoscopies can be greatly facilitated by performing them through a tracheotomic fistula of sufficient size to permit an easy introduction and passage of the bronchoscope. Were there repeated operations to be carried out through the larynx, the latter would in a very short time so react to the passage of the bronchoscope as to necessitate a tracheotomy for laryngeal obstruction alone. Moreover, a tracheotomic bronchoscopy is far less distress-

ing to the patient and far easier for the operator than when done through the larynx. Needless to say, no anesthesia is required or desirable in the face of such acute pulmonary infection.

From the moment when it becomes evident that by means of bronchoscopy an open airway can at least temporarily be maintained beyond the point at which inspissated crusts tend to accumulate there begins a desperate struggle to maintain and enhance the patient's resistance to a degree sufficient to handle the infection per se. In far the greater percentage of our patients who developed this fulminating type of infection, this was found to be impossible. Most of them lived only a few days, the infection spread downward, temperature, pulse and respiration rates all rose rapidly, and death ensued in spite of all measures employed. The use of an oxygen tent under such circumstances has proved useless and only large doses of morphin have been able to alleviate the sufferings of both the patient and parents.

In the rare instances where success has finally been achieved and the patient has been carried along by repeated bronchoscopies and by the most vigorous efforts to maintain a high fluid intake by means of fluids by mouth, intravenous injections and hypodermoclyses, it has proved to be a most prolonged and tedious procedure. The temperature, pulse and respiration rate have remained elevated for many days and only receded in a slow and gradual fashion. Periods would occur in which the patient was improved and apparently on the road to recovery, when out of a clear sky, and with no warning, bronchial occlusion would suddenly take place with a return of the typical dyspnea, again necessitating bronchoscopy. In the interim, repeated suction through the tracheal cannula with a catheter was necessary to remove the profuse purulent secretions produced within the tracheobronchial tree.

LITERATURE AND DISCUSSION.

Experiences with such desperate cases naturally leads one to inquire more deeply into the subject to discover, if possible, some more effective means of handling this situation and thereby of reducing the high mortality rate. Consideration of the literature on the subject at once discloses the fact that other laryngologists

have encountered this same clinical picture and in many instances have experienced the same disappointing and fatal outcome. Gardner,[1] in 1918, described three cases, all fatal, in which there was the typical symptom complex of fulminating laryngo-tracheo-bronchitis, together with evidence of acidosis. Baum, in 1924, reported four cases of the same disease, three of which died. All were treated by tracheotomy and all had the typical obstructing secretions in the trachea. Strachan,[2] in 1925, under the heading "Acute Septic Tracheitis," reported three cases, all of whom lived only three days. A most interesting case, reported by Clerf,[3] in 1924, described a foreign body case complicated by the same type of tracheitis in which it was necessary to perform seventeen bronchoscopies to remove obstructing crusts from the lower airways. Incidentally, it is interesting to note that several authors' cases which recovered not infrequently had a foreign body etiology with secondary development of tracheobronchitis. These cases were quite evidently more favorable than the purely infectious ones. Lynch,[4] in 1924, mentions having seen five of these cases in one month, all of which recovered, but in all of which he reports that the disease was practically afebrile. This is in direct contrast to all of the cases which we have seen in which a high initial febrile reaction was one of the outstanding characteristics.

In 1932 Gittins[5] reported his experiences with twenty-four cases of nondiphtheritic laryngitis and tracheobronchitis. The general clinical picture of these cases was approximately the same as that outlined above, the outstanding feature being the presence in the trachea of a thick, tenacious and crusting secretion, necessitating first of all tracheotomy and frequently later on bronchoscopy. His results were somewhat better than the general average, showing in the tracheotomized cases seven recoveries and eight deaths. In nine other cases which did not require tracheotomy, all but one recovered. Gittins stresses the importance of not overlooking the disease at the outset, by mistaking it for diphtheria, and hence of not relying on relief of the laryngeal obstruction alone, supplemented by administration of diphtheria antitoxin. Since the disease here under discussion never yields cultures of diphtheria bacilli, it is quite evident, as mentioned at

the outset, that an accurate diagnosis by laryngeal inspection, smear and cultures, must be made.

Other authors are in agreement that the infecting organism in fulminating tracheobronchitis is the streptococcus. It is this organism which can be obtained from tissue culture of the lung at postmortems. On the other hand, it has been frequently noted that cultures of the exudate attached to the subglottic walls and of that found in the trachea show staphylococci or pneumococci. These organisms must be regarded as contaminants or secondary invaders, but may play a most important part in the tendency for this pyogenic exudate to form the sticky, gummy crusts which are such a devastating factor in the production of lower respiratory obstruction.

The most recent article on this subject by Johnson[6] reports three cases, all of them under 3 years of age, all of whom recovered and on all of whom decannulation was accomplished within twelve days. Such rapidity of convalescence has, unfortunately, not been our experience in tracheotomized patients.

From the above brief accounts of the findings of other authors, it is quite evident that one is dealing, in fulminating laryngo-tracheo-bronchitis, with a condition of the utmost gravity, with one associated in most men's hands with a high mortality rate and one in which early and accurate diagnosis, followed by most intelligent and energetic treatment, can at best save only about half of the cases. To the results above summarized we wish to add a group of eleven cases, seen at the Children's Hospital in Boston, which were treated by a variety of procedures and which have led us to certain conclusions as to the most effective means as yet available for handling the condition. Our results may be briefly summarized as follows:

TABLE I.

Form of Treatment	Died	Cured
Tracheotomy	1 1 1 1 1	0
Tracheotomy and intubation	1	0
Intubation alone	1	1 1 1 1

The average age of these patients was 4 years, but the cases requiring tracheotomy fell within a narrow range between 18 months and 30 months. It is in this younger group of patients

that one may expect his most serious cases and his highest mortality. So often the onset of the infection is mistaken for simple spasmodic croup and treated as such, or by the administration of diphtheria antitoxin, that valuable time is lost during the first forty-eight hours, with the result that the endoscopist only first sees the patient when urgent dyspnea demands relief and tracheotomy is a matter of immediate necessity.

Something should be said here, before dismissing it, of the procedure of intubation, as applied to these cases. Most of the authors cited above specifically mention intubation in order to condemn it. It will be noted in the table of results of our eleven cases that four out of five treated by intubation recovered. On this basis one might at first justifiably conclude that a procedure which appears to be 80 per cent successful, even in such a small series, is worthy of at least some consideration. It must, however, be recognized that these cases in which intubation alone was successful were undoubtedly of a milder nature and did not tend to the downward extension of infection that was so typical of the other patients. In fact, one might be criticised for including them in any series of cases of fulminating laryngotracheitis since they did not develop tracheal extension. They are here included simply because at the outset, when direct laryngoscopy was done, they could not be distinguished in any way from the more fulminating type of case. They had the same dry crusting of the subglottic walls, yielding staphylococcus, were entirely unlike diphtheria, grossly and by culture, and had the same need for the relief of laryngeal obstruction. Intubation was done, always with the possibility that it might prove inadequate and that tracheotomy might be necessary later, as in the two combined cases it did. If intubation alone will prove adequate, it is a simpler procedure than tracheotomy. If not, it had best be omitted and tracheotomy alone performed. One reason for this is that the presence of an intubation tube in an already infected subglottic region only further irritates the space and tends to prolong the process of decannulation. Nothing is more aggravating in these cases which have survived a combined intubation and tracheotomy than to find that in spite of a low opening and subsidence of active tracheal infection the tracheal tube cannot be

removed at the end of a normal period of proper decannulation. For this reason alone, if tracheotomy is at all anticipated, intubation should be omitted. Furthermore, the dangers of the tube being coughed up are ever present, necessitating the constant presence of someone capable of replacing the tube. Two or more such replacements will almost certainly necessitate a tracheotomy before extubation may be successfully accomplished.

A further disadvantage of intubation is the difficulty encountered, especially with infants, in inducing the intubated patient to take fluids by mouth. One or two fits of coughing occasioned by fluids going down the tube are sufficient to induce most children to resist any further attempts to force their fluids. Since no one therapeutic measure is so important, aside from mechanical relief of the obstruction as to promote a more than adequate fluid intake, it is at once apparent that intubation is to be avoided on this ground alone if for no other. One thing, however, should be made clear before dismissing intubation. Its disadvantage does not lie primarily in the fact that it will not relieve laryngeal obstruction as effectively for the moment as tracheotomy. In our experience it does this quite efficiently, until the formation of lower tracheal secretions and obstructing crusts makes it, as well as tracheotomy, ineffective. On its removal under these circumstances the tube will be found to be patent and the secretions more fluid in character than those that tend to plug a tracheal cannula. This can only be due to the fact that warm, moistened air still passes through the nose into the tube, avoiding the drying effect incident to the tracheal cannula. Finally, if tracheal infection develops, with its attending obstruction, tracheotomy has an overwhelming advantage over intubation, not in relieving the laryngeal obstruction, but in permitting frequent bronchoscopic removal of tracheal and bronchial plugs in the manner to be subsequently described.

On the other hand, a recent article by Baum[7] stresses the advantages occurring from replacing the tracheotomy tube by an intubation tube in order to avoid the tendency to crust formation incident to the inspiration of dry, unwarmed air through the tracheal tube. Baum feels that this is an important etiologic factor in the bronchial crust formation and hence should be avoided by

resort to the intubation tube. Personally we have felt it so helpful to utilize the tracheal wound in the bronchoscopic approach to the lower bronchi that we have hesitated to return to the intubation tube where it had previously proved inadequate. Moreover, if the intubation tube is to remain in place for any appreciable time, there would seem to be a considerable risk of laryngeal ulceration and subsequent stenosis. Baum, however, has reported favorable results following the adoption of this change from tracheotomy to intubation.

The mild type of infective, subglottic infection which in our experience can sometimes be successfully treated by intubation is illustrated by the following case. In any but such mild cases it should not be resorted to.

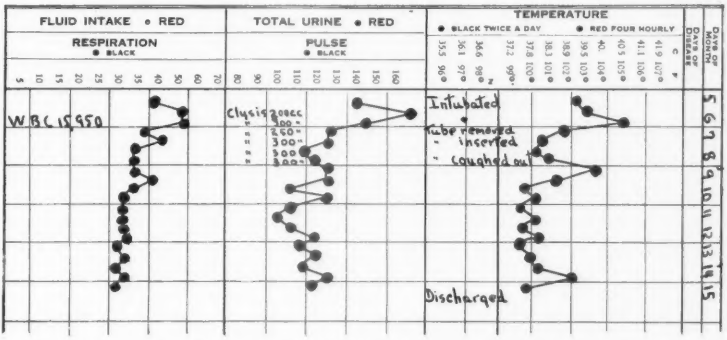

Fig. 1. Case 1. Mild form of infectious laryngeal obstruction, treated by intubation alone.

Case 1.—M. M., aged 1½ years. Admitted January 5, 1932; discharged January 16, 1932. (Fig. 1.)

P. I.—This patient was admitted because of difficulty in respirations following an acute infection. Child showed signs of laryngeal obstruction.

January 5, 1932. Operation.—Inspection of the larynx showed dried crusts on both cords. This crusting was removed and an intubation tube inserted.

January 6, 1932. Following intubation, patient was kept in croup tent and benzoin inhalations given. Very much more comfortable. Some mucus in throat but child slept in naps. Given clysis of normal saline. There was some difficulty in getting patient to take liquids.

January 7, 1932. Operation.—Without anesthesia, the intubation tube was removed. Following this respirations became more labored and child

refused fluids. Child was taken to the operating room and the tube reinserted and another clysis given. Child then breathed noisily but with ease.

January 8, 1932. Child spoke for the first time today. Still refuses fluids. Another clysis was given and child slept well but refused all fluids until 6 a. m. Respirations labored.

January 10, 1932. Child bright and happpy. From this time on patient had no particular difficulty except occasional coughing spells. Temperature fell to normal and patient was discharged January 16, 1932, improved.

Laboratory data: At time of admission white count was 15,950. Mucus from larynx on culture showed pneumococcus and staphylococcus aureus.

Comment.—It is quite possible that such a case as the above might have responded equally as well to simple laryngoscopic, mechanical removal of the accumulated secretions from the glottic wall, without the use of an intubating tube, even if this procedure of removal had to be repeated on several occasions. This is a form of treatment which has proved quite efficacious in the treatment of laryngeal diphtheria. Taking into account the relative merits and disadvantages of intubation one cannot but conclude that it is not the procedure of choice in the treatment of any acute laryngeal infection which shows any likelihood of developing further downward extension into the trachea.

What happens when intubation alone is relied upon in the presence of lower tracheal obstruction is well illustrated by case 2. This was one of our first patients whom we observed with this fulminating type of infection, and is reported only to show the hopeless inefficacy of trying to relieve the laryngeal obstruction without dealing more directly with the bronchial occlusions below. True, attempts were made to aspirate the trachea through the larynx on repeated occasions, but this does not begin to compare with direct vision removal of occluding crusts through the bronchoscope. It is doubtful if even with tracheotomy this child, so desperately ill on arrival, would have recovered under any form of treatment, but tracheotomic bronchoscopy would unquestionably have been the procedure of choice.

Case 2.—E. W., aged 2¾ years. Admitted November 10, 1930; discharged November 11, 1930. (Fig. 2.)

C. C.—Brought to the hospital at noon because of marked difficulty in getting breath and cyanosis for about two hours. Parents unable to reach family physician.

P. I.—Eight days ago; gradual onset of hoarseness. Other two children had colds at this time.

Five days ago: Cough and hoarseness worse, in bed. Then gradually worse. In croup tent.

One day ago: Slight difficulty in getting breath; more noisy.

Today: Suddenly very blue, extremely restless, noisy breathing.

P. E.—Critically ill. Extreme cyanosis. Respirations labored, rapid, regular, marked supra- and infra-sternal retraction on inspiration. Breath foul with slight acetone odor. Thick, purulent discharge from right nostril, slight from left. Mouth dry. Throat red. Lungs resonant throughout, coarse, moist rales medially. Heart rapid, abdomen and extremities negative.

Course: Direct laryngoscopy revealed some edema of the larynx and a trachea almost filled with thick yellow tenacious exudate, but no marked edema. The exudate was removed by suction and the larynx intubated. Smear of the laryngeal exudate showed diplococci, morphologically pneumococci. No K. L. bacilli. The patient's color improved remarkably. Then given saline solution subcutaneously and 10 per cent glucose solution intravenously. Coughed tube up two hours later and was reintubated by direct laryngoscopy. Color then remained relatively good until suddenly, about 6:30 p. m., patient gradually became very restless and extremely

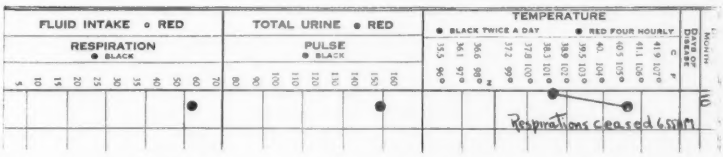

Fig. 2. Chart of Case 2, showing rapid demise of patient.

cyanotic. Laryngoscopy again performed with suction and reintubation. At this time showers of petechiæ appeared about face, neck and extremities. The petechiæ were thought to be probably due to asphyxiation. Temperature remained elevated, respirations rapid and child almost constantly cyanotic, relieved to some extent by frequent suction, applied to bronchi and trachea through direct laryngoscopy. During the early morning hours he gradually became worse and died at 6:15 a. m., eighteen hours after admission. A limited postmortem revealed extensive inflammatory reaction, involving the larynx, trachea and bronchi, with some patchy congestion in the lungs. Many of the smaller bronchi were filled with thick, tenacious, mucopurulent material. Cultures of this material showed both hemolytic streptococcus and pneumococcus.

Diagnosis: Laryngeal tracheobronchitis. Bronchopneumonia.

X-ray report: November 10, 1930. A film of the chest shows a considerable amount of hazy congestion extending out from the hili, particularly on the right, and due evidently to a recent severe bronchitis which on the right amounts to practically early bronchopneumonia. There is a tube in the upper trachea.

Comment.—No written description can adequately describe the severity of illness in such a case. There appears to be a combined effect of intense toxemia coupled with recurring respiratory obstruction. Undoubtedly relief of the latter will accomplish much, and if the patient's resistance can cope with the toxic element, recovery will ensue in certain cases. In others, there would appear to be such extensive pulmonary pathology as to absolutely preclude any chance of recovery. What these pathologic changes are will be discussed below in the report of the pathologist.

We come now to a consideration of tracheotomy as the procedure of choice in dealing with these cases. As has so often been emphasized by Jackson, the procedure should be done early; its postponement only renders the operation more difficult and likewise subjects the patient to increasingly inadequate oxygenation during a period when he most needs it. The ideal arrangement is that in which direct laryngoscopy definitely affords a diagnosis of nondiphtheritic, descending laryngotracheitis. If this is followed immediately by bronchoscopy, two great advantages are achieved. First, the lower trachea can be examined and any obstructive secretions or crusts removed, with the result that one knows positively that at least at this stage the lower airway is free. Moreover, the bronchial orifices can be examined and some idea obtained as to the severity of reaction present and the degree to which the bronchi share in this. Secondly, and of equal importance, the bronchoscope can be left in place and the tracheotomy performed tranquilly with the tube as a guide and with entire assurance that there will be no unpleasant developments of laryngeal obstruction during the operation. Once one has performed tracheotomy in the presence of severe laryngeal obstruction by this method he will resort to it more and more frequently with increasing satisfaction.

There is some difference of opinion as to the type of incision to be made into the trachea. Needless to say, the incision should always be "low," through the third or fourth tracheal ring. Only by maintaining this rule can one avoid the distressing results manifested by difficult and prolonged decannulation, which attend a "high" tracheotomy, even if this does not in any way injure

the cricoid cartilage. Any encroachment on the subglottic region, particularly in a young child, will almost surely give rise to trouble. In any case, where no further tracheal or bronchial manipulation is anticipated, and where the only requirement is the relief of the laryngeal obstruction, a simple linear or crucial incision in the trachea is quite sufficient. Since, however, in the type of case here under discussion, repeated tracheoscopies and bronchoscopies will be necessary to maintain an adequate airway during the period of acute infection, we have come to feel that a modified tracheal opening is of great help. This modification consists of a crucial incision with the removal of small portions of the four corners, leaving a circular opening in the trachea (the so-called punch tracheotomy of Mosher). It is realized that this procedure may be open to the criticism that such a defect will not heal as readily and that there may be some tendency for the ingrowth into the tracheal lumen of granulation tissue. In our cases so treated, however, there has been no definite evidence of this disadvantage and in experimental work done by the author on cats this type of tracheal opening did not leave any evidence of tracheal stenosis.

Our reason for preferring this circular tracheal opening has been that it so greatly facilitated the introduction of the bronchoscope in subsequent bronchoscopies. Where one is faced with the possibility of repeated urgent bronchoscopies, for as many as seventeen times, we have found that introduction through the tracheotomic wound was easier for the operator and immeasurably easier for the patient. We have felt that repeated bronchoscopies through the larynx, in the presence of infection, could easily result in some laryngeal edema, if not actual trauma, which might render subsequent decannulation more difficult than if the larynx were left undisturbed. Tracheotomy is certainly not indicated for any ordinary bronchoscopy, but where the latter procedure may require frequent repetition the lower route has proved more satisfactory in our hands. We mention this, even in the face of Jackson's statement that this procedure has been "rendered obsolete" and that "it is easier to work with a bronchoscope through the mouth than through the neck."

Once a properly fitting cannula of adequate size has been inserted, the immediate difficulty of laryngeal obstruction has been relieved. From this moment the endoscopist must plan a campaign requiring all the strategy of a military general in battle in an endeavor to forestall the development of any condition deleterious to the recovery of the patient. The latter should be placed in a warm room, the air of which is kept constantly moistened by some form of artificial humidifier. Whether the addition of such medication as compound tincture of benzoin is of value is questionable. Special nurses, day and night, should be graduates, thoroughly familiar with the care of tracheotomized patients and particularly on the alert for the slightest suggestion or evidence of any increased difficulty in breathing. We can conceive of no condition in which careful and intelligent nursing can play such an essential and important rôle as in the care of these cases of fulminating tracheobronchitis. Perfectly functioning suction apparatus must be at the bedside, to which a sterile rubber catheter is attachable. At the least sign of accumulation of tracheal secretion the inner cannula should be removed and the catheter inserted into the trachea. This will usually take care of fluid material in the upper trachea. It will not, however, remove the sticky, glue-like crusts which are such a dread factor in this disease. In our experience, these can only be removed by the aid of bronchoscopy, since they are so likely to lie below the bifurcation and beyond the reach of any form of indirect suction. For this reason it is essential for the endoscopist or his assistant to remain within a few moments' call and for the operating room to be ready at a moment's notice for the carrying out of this procedure. A combination of bronchoscopic suction and the use of foreign body forceps will usually serve to remove the occluding crusts and restore the airway.

Without some experience with these cases, one can hardly realize the suddenness with which this tracheal occlusion can take place. A child may seem to be breathing comfortably and relatively slowly and to have a free airway when there will in a few moments occur a most profound change in the whole general condition. Restlessness, a rise in the respiratory rate from, say 40 up to 60, a great increase in the audibility of respiration, the

development of marked substernal retraction, a rise in pulse rate and, above all, a peculiar change in the patient's general appearance—all these are unquestionable evidences that the airway in the lower bronchi is becoming rapidly occluded and that relief is urgent. If in the meantime suction with the catheter through the tube does not afford any relief, immediate bronchoscopy is the only adequate procedure. Many authors have stressed the folly of attributing such symptoms as these to the development of pneumonia. Subsequent terminal bronchopneumonia may well occur in those patients in whom the general septic process becomes too extensive and overwhelming for the patient to overcome, and in such cases postmortem examination will inevitably show lung consolidation, but with the picture of rapidly increasing dyspnea in a patient tracheotomized for infectious laryngeal obstruction all the therapeutic measures should be directed to the immediate investigation and relief of this obstruction. Second only in importance to maintenance of an open airway is the adequate administration of fluids. Whether given by mouth or by hypodermoclysis, or intravenously, it is highly essential that these patients be given a more than normal amount of fluids. A persistent high fever, an open tracheal fistula and the general tendency to crust formation in the upper airways make it very necessary that the tracheal secretions be made as profuse and as liquid in character as possible. No expectorant will be of any avail unless the tracheal secretions are present in adequate amounts, and the first hopeful sign in any of these cases is the appearance of a more normal tracheal secretion with a loosening of the former viscosity. During the height of symptoms, aside from all possible fluids given daily by mouth, we feel that at least 500 cc. of saline should be given under the skin to a 2-year-old child, and that intravenous glucose (300 cc.) should be given each day for the first three days. The tendency for these patients to develop acidosis only aggravates this respiratory infection, and measures designed to combat this will be equally valuable in overcoming the latter.

Whether any specific solvent of this glue-like tracheal secretion can be utilized is still a question. We felt that sodium bicarbonate solution, instilled regularly into the tracheal cannula seemed to soften secretions and, combined with catheter suction, made it

easier to aspirate them. In view of the frequent finding of staphylococcus on smearing the secretions, we employed staphylococcus bacteriophage in the same manner, but without definite evidence of its value. Gittins strongly recommends the use of adrenalin in this manner.

Finally, the use of antistreptococcus serum has been tried. If this infection is primarily and fundamentally a streptococcus one, this serum would seem to be a most valuable and reasonable therapeutic measure were it not for the specificity of the organism and the difficulty in finding a serum which will match the strain of the particular streptococcus in any given case. Whenever this becomes possible, we shall have gone further in the treatment of these desperate cases than it now appears possible to go.

Under the heading "Clinical History of a Typical Case of Acute Infective Laryngotracheitis," Jackson[8] ascribes the etiology to the influenzal bacillus. In our cases and those of most other authors, this organism has not been found and, except in such influenzal epidemics as have occurred in the past, the streptococcus would seem to be the common etiologic organism. It is quite true, however, that in the cases coming to postmortem the lung changes were frequently almost identical with those found so commonly during the influenza epidemic of 1918. Similar cases occurring during the influenza epidemic were reported by Meyer and also by Stettner in Germany. Since the streptococcus was so commonly found in the lungs of patients who had died from influenza, it is possible that the streptococcus may have been responsible for the disease picture described by these authors, and that the diagnosis was made more difficult because of the occurrence of these cases during a huge influenza epidemic.

The high mortality in our own cases and those of several other authors has been mentioned. Preliminary experience with unsuccessful cases always leads one to hope that, profiting by such experience, he may be more successful in subsequent occasions. We wish, therefore, in some detail to record our most recent case, because it exemplifies the carrying out of all the procedures outlined above, and shows how, by maintaining a constantly clear airway and by attention to adequate fluid administration, even a most fulminating infection can be overcome.

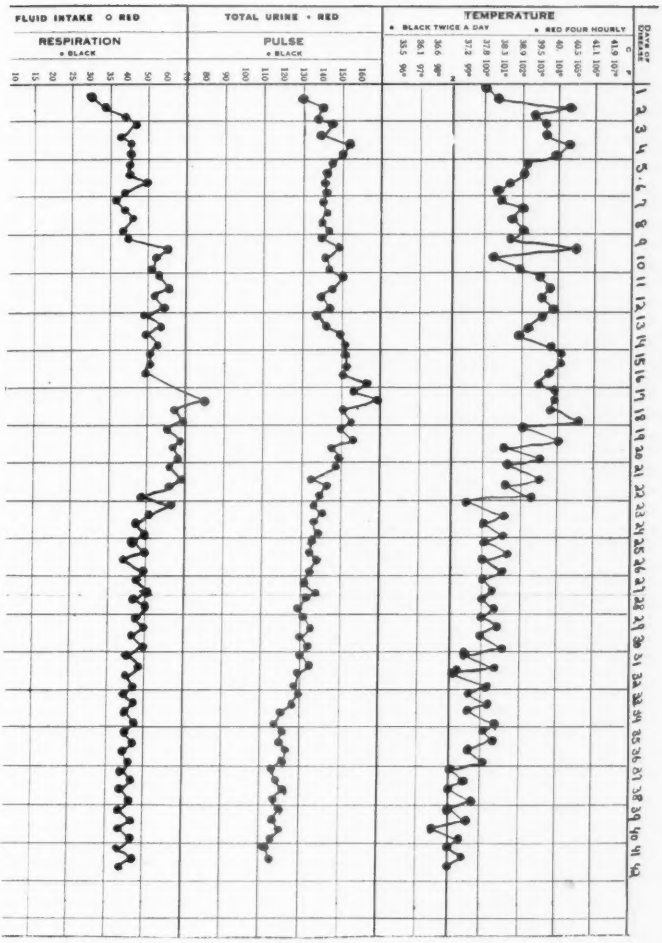

Fig. 3. Temperature chart of Case 3.

Case 3.—B. K., aged 3 years. Admitted March 18, 1932; discharged May 14, 1932. (Fig. 3.)

P. I.—This child had a history of having been perfectly well until five days ago, when she awoke with a croupy cough. For the next twenty-four hours she did not seem particularly sick except for the cough. Tem-

perature was not essentially elevated. On Friday, March 18, she seemed to be worse and a pediatrician was called in the morning, who thought she would improve, but advised hospitalization.

March 18, 1932. When seen at noontime patient looked extremely ill, having marked substernal retraction, cyanosis and great difficulty in breathing. Examination of the larynx showed the cords to be normal in appearance, without redness or swelling. Looking between the cords one could see typical crusts, adherent to the walls of the trachea. These were removed with forceps and suction. A 2-year-old intubation tube was inserted with great relief to the dyspnea. Throughout the rest of the day child seemed comfortable, got free exchange of air through the tube, took fluids by mouth fairly well. Temperature, pulse and respirations continued to rise. She went through Friday night without difficulty.

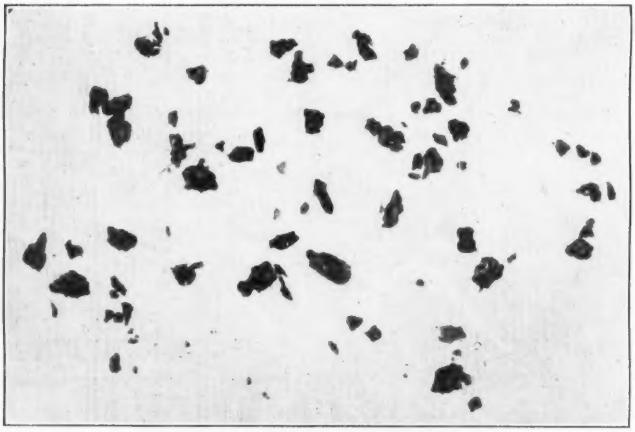

Fig. 4. Group of inspissated bronchial secretions removed by bronchoscopy from Case 3.

Comment.—Up to this point the case resembled in almost every particular those others in which prompt recovery followed intubation (see case 1). The subsequent course, however, indicates how useless intubation can be, and in this instance it would have been as well to have omitted it and proceeded directly with tracheotomy. The progress notes are as follows:

March 19, 1932. During Saturday patient was fairly comfortable except for certain periods of marked restlessness, not particularly due to dyspnea. X-ray of the chest showed nothing abnormal and except for the fever of 103 degrees and high pulse rate, she had a fairly satisfactory day and night.

March 20, 1932. Bronchoscopy and Tracheotomy.—The intubation tube was removed and was not found to be plugged, but the walls of the larynx showed numerous areas of mucoid deposit as did the upper portion of the trachea. A 4 mm. bronchoscope was easily passed through the cords down to the bifurcation of the bronchi and secretions were sucked out. Since this procedure resulted in marked improvement in the patient's condition, return of good color and quiet respirations, it seemed quite obvious that a tracheotomy was advisable. This was done with the bronchoscope in place. Punch tracheotomy was performed, leaving a large window in the anterior tracheal wall. A No. 2 tracheotomy tube was inserted and the patient returned to bed in a comfortable condition.

March 20, 1932. Child continued to breathe well and be comfortable; her color was good, but her temperature continued to rise. She took fluids fairly well by mouth and did not appear dehydrated.

Comment.—The failure of temperature and pulse rate to fall is indication of further downward extension of the infection.

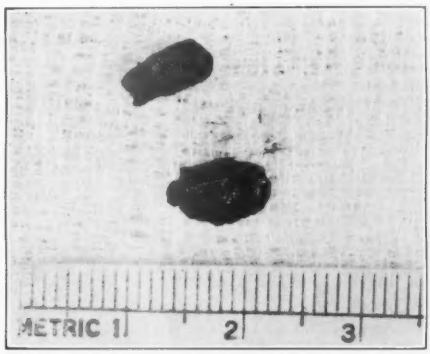

Fig. 5. View showing size of crusts seen in Fig. 4.

March 21, 1932. At 2 a. m. this morning her cyanosis returned and by 3:30 a. m. she was extremely cyanotic and was in very poor condition. She was immediately taken to the operating room and a bronchoscopy performed.

A 4 mm. bronchoscope was inserted through the tracheotomy wound and both main bronchi found to be plugged with tenacious material. This was removed with forceps and suction with marked improvement in the child's condition.

March 21, 1932. Child continued to breathe well until 11 a. m. this morning, when the same situation returned. She was again taken to the operating room and bronchoscopy performed and tenacious material removed. Her condition did not improve quite as much and her color was dusky. Her pulse and temperature continue high. Subcutaneous saline

and intravenous glucose given early this morning. The patient continued in about the same condition during the remainder of the day. Early in the evening the use of staphylococcus bacteriophage was begun, using it with a medicine dropper, dropping one-quarter full dropper down the trachea every half hour, with the hope that this might soften up the sticky secretions. During the late afternoon and evening the child's condition was better, and though pulse and temperature were still high her color remained good and she took fluids well. At 10 p. m., although she was in good condition, she was again bronchoscoped and several large masses of tenacious material were removed. I had the feeling that the crusts were somewhat less dried out and the bronchial walls on both sides less swollen. On each side one could see down the main bronchi to the lower subdivisions. These were quite clean-cut and appeared almost normal. Child went through the night without any difficulty except for some coughing.

March 23, 1932. Child went through the night until 5 this morning, and then almost within five minutes her condition changed from a satisfactory to a desperate one. Her color became blue, pulse poor, respirations rapid and she was extremely restless and fighting for air. She was again bronchoscoped and a number of dried crusts removed from both bronchi. General picture was that of increased dryness in secretion and more inflammation of the walls. Following bronchoscopy the condition improved considerably, but her temperature, which had fallen to 101.5°, rose to 103°. She was given 300 cc. saline under the skin and seemed quite exhausted by this flare-up, although it was quite amazing to see the relatively normal attitude she maintained toward her environment in the manner of drinking fluids, playing with her toys and responding with clear mentality to all questions asked. With the increase of dried secretions and the rising pulse and temperature, the prognosis seemed less hopeful. Patient had taken large amounts of fluid, amounting to almost 60 ounces, plus 950 cc. salt solution under the skin and 350 cc. of glucose intravenously. The use of staphylococcus bacteriophage was discontinued and a solution of soda bicarbonate, dropped down through the tracheotomy tube, as it seemed to be more or less solvent to the secretion.

Temperature and pulse remained about the same, once going down to 101.4°. It gradually became possible to suck out pieces of secretion by the use of a catheter introduced through the tracheotomy tube. Patient entered the eighth day of her illness with everyone feeling more encouraged about her. The night was passed without any acute flare-up or development of obstruction. The tracheotomy tube was frequently sucked out and masses of secretion still came up. Her color was better.

March 24, 1932. This morning patient seemed fairly comfortable in spite of her serum sickness. Temperature and pulse higher but she was certainly no worse than yesterday.

March 26, 1932. This child did not seem qiute so well today. About 4 o'clock her bronchi were aspirated and several crusts removed and she seemed quite comfortable. The respirations were somewhat rapid with elevation of the pulse and temperature.

March 27, 1932. Today the child did not seem quite so well. Nothing outstanding except for rapid respirations and pulse and a tendency for the temperature to continue to rise. She had a fairly comfortable night and

the same treatment has been continued. Bronchoscopic examination and aspiration yesterday afternoon yielded a few small crusts but no sticky masses as heretofore. The child seemed to be losing ground. The color is fairly good. She was alert but restless.

March 28, 1932. A fairly good night last night. This morning her temperature was high and reached a peak of 105°. The pulse had gone to 170. X-rays of the chest showed no gross pneumonia, but merely accentuation of the peribronchial markings. By suction it was still possible to remove thick yellow secretions and the child herself did not look as badly as her chart would indicate.

March 29, 1932. Fourteenth day of illness. More or less thick secretions from the chest still aspirated but not quite as much plugging up with dry masses as last week. Pulse, temperature and respirations continued high without any abatement except occasional drop in temperature to 101° or 102°. On various occasions patient showed remarkable interest in her surroundings, even sitting up to look at picture books. She was quite capable of holding her own glass when taking fluids. About 6 o'clock tonight bronchoscopy, because of more rapid respirations, revealed one hard mass in the left bronchus and also a few small ones. Bronchial mucous membrane below these points did not look as thick as heretofore, but purulent secretion was seen coming from below. Child's condition unchanged during the last forty-eight hours.

March 30, 1932. Last night patient had a fairly comfortable night; slept in naps. She was given supplementary fluids in her skin and she took about three quarts of fluid by mouth yesterday. Pulse, respiration and temperature still high. Color fairly good. About 4 o'clock this afternoon this patient's breathing was rather more rapid in spite of her condition seeming about the same and she was bronchoscoped again. There were no crusts in the main bronchi and only a purulent secretion on each side. The bronchial lumen on both sides seemed to be less patulous than on previous examinations.

March 31, 1932. This morning, child's condition was not so good. Eight o'clock temperature was 105.° Her skin was pale and her lips slightly dusky. Four hundred fifty cc. clysis was given, which she took readily, resulting in slight improvement in her appearance. Pulse and respiration remain unchanged. Patient passed a fairly uneventful day without much change. Another clysis was given in the afternoon. She had a fairly comfortable night. Pulse and respirations remain high. There has been relatively little change during the night.

April 1, 1932. Child's condition remained unchanged, and it was not necessary to perform another bronchoscopy. Temperature, pulse and respirations remained high. Took ample amount of fluid supplemented by clysis.

April 3, 1932. Patient seemed alternately to look fairly well and then very badly. Pulse and respirations and temperature still up. Temperature fluctuated from 101-102 to 105 degrees. Bronchoscopic suction nearly every day.

April 4, 1932. This morning patient looked fairly well. In the afternoon she looked poorly, was cyanotic; temperature rose to 105, respirations

to 70 and pulse to 180. Fatal outcome seemed inevitable. She had, however, a fairly good night.

April 6, 1932. There has been a decided break in the character of her temperature, which is more picket fenced in type but receding somewhat. Pulse and respirations are lower. Bronchoscopy not necessary today.

April 9, 1932. Bronchoscopy today showed essentially the same condition. More pus was seen in the right bronchus than the left. The bronchial lumen appeared somewhat narrowed. It seemed worth while to see what effect the oxygen tent would have upon her, and she was placed in it, appearing more comfortable for the following twenty-four hours, with some lowering of the respiratory and pulse rate. Her color was improved.

April 12, 1932. Patient's temperature was a shade lower but coming down very, very slowly. In the oxygen tent most of the time, color being better when she is in it than when she is out of it. Large amounts of purulent secretion are being aspirated with a catheter and also a large number of crusts have been brought up or coughed up through the tube. The falling temperature and slight drop in pulse and respirations are encouraging.

April 21, 1932. Condition about the same. Slight improvement from the clinical standpoint manifested by her interest in things and her desire to play with toys. Continued to have pus and secretions and still some crusts removed by suction.

May 6, 1932. Continues to improve. Mucopurulent secretions still present and must be removed from her trachea. Still occasional crusts removed in this way. She has learned to cover her own tracheotomy tube and talks quite freely in this manner.

May 14, 1932. Died suddenly from unknown cause.

Comment.—This little girl had been making what everyone who saw her regarded as a steady and rapid convalescence and had had a normal temperature for ten days up to the day of her sudden death. This occurred entirely without warning. The patient suddenly turned pale, and died before anything could be done. Subsequent bronchoscopy revealed a perfectly patent airway but even in a Drinker respirator breathing could not be induced. A similar delayed fatality, unexplained even at autopsy, was reported by Baum in 1928. In spite of this outcome after fifty-seven days of illness, we feel that the initial tracheobronchial infection had been essentially overcome, a feeling borne out by postmortem findings (see below). We are convinced that in such desperately ill patients it is only by pursuing some such course as was followed in the case of this patient that life can be saved and the patient restored to health.

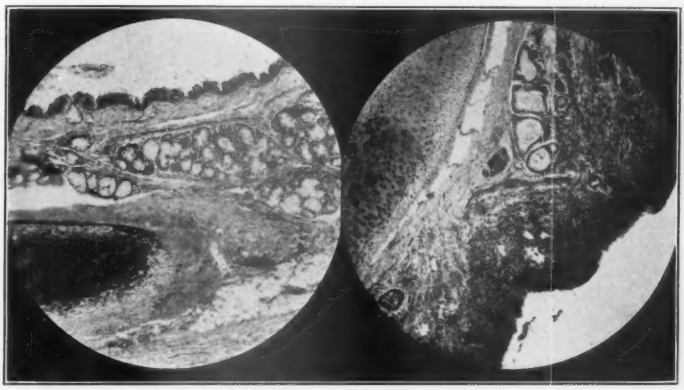

Fig. 6. Section showing normal tracheal mucosa, for comparison with Figs. 7, and 10 (a), and 11.

Fig. 7. Photomicrograph, showing the intense inflammatory reaction in the tracheal mucosa of Case 2.

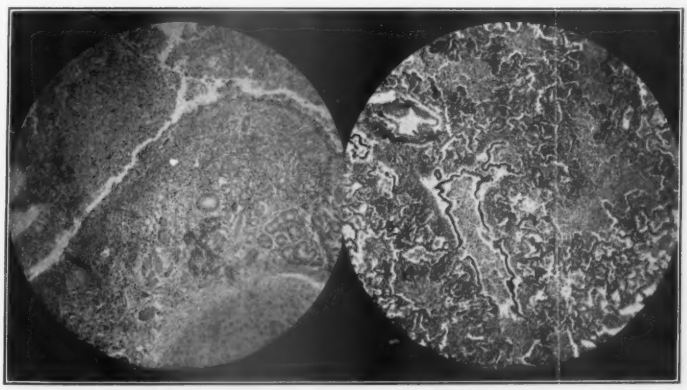

Fig. 8. Bronchial wall of Case 2, showing intense inflammatory reaction.

Fig. 9. Lung, in Case 2, showing diffuse bronchopneumonia and a small bronchiole, filled with purulent exudate.

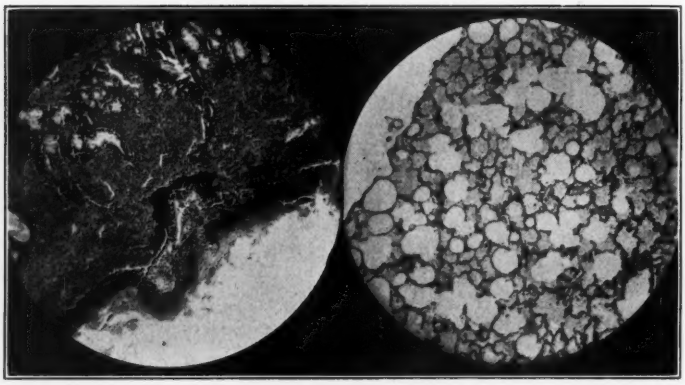

Fig. 10 (a). Marked destruction of tracheal mucosa from case of acute tracheobronchitis.

Fig. 10 (b). Lung from same case shown in Fig. 10 (a). In spite of intense tracheal inflammation, the lung at the same time shows relatively little pathology.

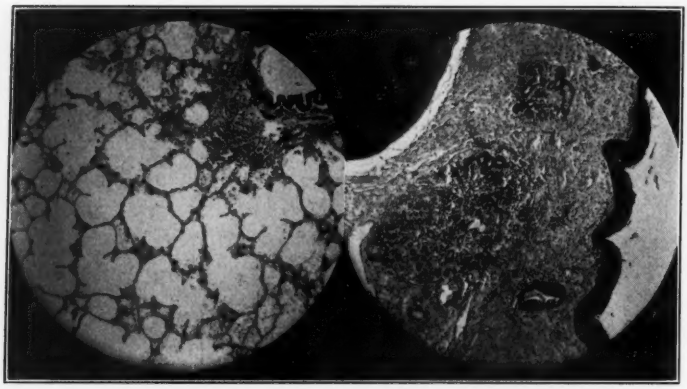

Fig. 11. Lung and small bronchus from Case 3. No pathology beyond some peribronchial inflammation.

Fig. 12. Section of tracheal mucosa from Case 3, 54 days after onset of illness. Note healed epithelium with change from columnar ciliated to squamous stratified type.

PATHOLOGY.*

We have been particularly interested in the pathologic changes which occur in this disease, in the trachea, bronchi and lungs. We have been able to study these in a number of patients, and have selected for presentation here sections from case No. 2, a rapidly fatal one, and case No. 3, on which recovery, from an infectious standpoint, had taken place. The pathologic findings in these cases present one of the variations of damage caused by invasion of the upper respiratory tract by streptococcus hemolyticus. That organism was obtained in pure culture at autopsy in all instances, with the staphylococcus and the pneumococcus as occasional secondary invaders. The changes are essentially those of an acute descending inflammation of the upper respiratory tract, with the extent of involvement dependent upon the duration of life after onset of symptoms. The most striking change was the diffuse acute cellular infiltration and destruction of the walls of the larynx, trachea and bronchi. The histologic picture is comparable to that of a cellulitis of the skin, and is characteristic of the streptococcus. A second type of lesion was seen in the destruction of the mucosa and the masking of the mucosal surfaces by fibrin and purulent exudate. In those cases where a superimposed infection with the staphylococcus occurred, a more marked destruction of the mucosa and wall of the trachea could be noted, as a manifestation of the necrotizing characteristics of the staphylococcus.

In the lungs there was considerable variation in the degree but not in the type of the inflammatory reaction. The important findings were the peribronchial reaction and the polymorphonuclear infiltration of the alveolar walls due to the invasion of the streptococcus. In several instances following vascular injury a terminal spread to the alveolar spaces and bronchiolar lumina had occurred, with the formation of a true bronchopneumonia.

The autopsy findings in the case of the child who had lived for fifty-four days following the onset of symptoms showed a some-

*The pathologic examinations in these cases were made, and the accounts of the microscopic findings here given were written, by Dr. Sidney Farber, pathologist to the Children's Hospital, Boston, Mass.

what different picture, although the basic changes remained the same as in the others of this series. Practically all trace of acute inflammation had disappeared. The destroyed mucosa of the trachea had been repaired and, interestingly enough, replaced by a stratified squamous type of epithelium. Such metaplasia of epithelium is sometimes associated with the repair following continued severe inflammatory damage. There was no evidence of pneumonia. Only a slight peribronchial infiltration of lymphocytes and large mononuclear cells gave evidence of the previous extensive infiltration. From the autopsy findings it must be concluded that the streptococcus infection had ceased, that repair was taking place, and that no active acute inflammation of the upper and lower respiratory tract existed at the time of death. The sudden exitus can be best explained by the rupture of a sinus tract running from the wall of the trachea to the mediastinum. Mediastinal emphysema was definitely present and of severe enough degree to account for death.

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LXXXI.

A STUDY OF THE HYDROGEN-ION CONCENTRATION, NITROGEN CONTENT AND VISCOSITY OF NASAL SECRETIONS.

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ST. LOUIS

Although the importance of changes in the character of the secretions of the mucosa of the nasal cavity and accessory sinuses in diagnosis and treatment of upper respiratory diseases has long been appreciated by the clinician, it is only within the past few years that any chemical study of nasal secretions has been attempted. In 1930 Mittemaier[1] made a comprehensive study of the hydrogen ion concentration of normal secretions and secretions procured from cases of sinusitis. His normal or so-called "stoppage" secretions were obtained from cases of chronic rhinitis resulting from large adenoid swellings, hypertrophied turbinates or septum deviations. These were the simplest forms of chronic rhinitis in which there were no vascular, polypoidal, papillary or similar changes of the mucous membrane. In the seven cases studied the pH at 18° C. ranged from 7.57 to 8.41. Mittemaier, using the classification of Nübsman, divided sinus diseases into four groups, which are as follows: (1) Mucopus recurring, in which symptoms are temporarily relieved by irrigations but frequently recur; (2) purulent-fetid recurring, in which relapses are frequent and conditions rapidly improve following irrigation; (3) purulent-fetid constant, in which the secretion is fetid purulent and does not change in character and in which there is no response to treatment; (4) mucopus constant, an atrophic form, which changes neither quantitatively nor qualitatively to treatment. In groups one and two the pH increases as the cellular content of the secretion decreases. The pH of the secretions in cases of acute

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inflammatory processes is definitely on the acid side, whereas, in catarrhal conditions pH is on the alkaline side. Mittemaier suggests also that a constitutional factor plays an important role in the pH values of secretions in sinus affections. His findings, he asserts, are independent of chance happenings and that they represent a good picture of the nasal pathology.

Beckman[2] believed that potential alkalosis, which he defined as "the tendency of the individual to store up too much alkali reserve without necessarily becoming clinically alkalotic," is an important causative factor in allergy. In support of this view, he cited the rare occurrence of allergy in diabetics, a decline in allergy during starvation, the beneficial effect of therapeutic measures that tend to produce an acidosis, and the relief afforded patients afflicted with allergy during an acute infectious disease. He advocates mineral acid therapy in allergic maladies.

Fineberg[3] determined the carbon dioxide combining power of the blood in selected nasal conditions and divided them into six groups, namely: (1) Chronic purulent sinusitis, mucosa red; (2) hyperplastic sinusitis, no pus, mucosa red; (3) allergic rhinitis, mucoid, mucosa pale; (4) allergic rhinitis, no secretion, mucosa pale; (5) combined allergy and sinusitis; (6) acute rhinitis. In all conditions he found that the carbon dioxide combining power fell within the normal limits and that the beneficent effect of acid and alkalin therapy are not because of any direct relation to the acid-base equilibrium. He suggested that the benefit may be due to local changes when the attempt is made to change the acid-base equilibrium of the blood and also due to some factor, related to the acid-base equilibrium, but not reflected in the blood determinations.

Israel and Nicholas[4] have made an extensive quantitative study of the changes in blood, pus, tissue and bony wall of diseased sinuses in the hope of securing data of clinical value. They concluded that their finding did not warrant diagnosis, prognosis or treatment. Recently Mittemaier[5] presented a paper concerning the water and mineral content of nasal secretions. He concludes that the greatest increase in mineral content is found in the secretion of acute rhinitis; (2) in chronically inflamed sinuses, the

mineral value is high and remarkably constant; (3) sudden changes in the secretion, as from a purulent secretion to a mucous one, is accompanied by an increase in minerals; (4) edematous mucous membrane contains more mineral than the solid flat mucous membrane. The water content is greatest in the glassy polypose tissue and less in the flat, smooth mucous membrane. The water content of the secretions is very variable.

The physical-chemical study of secretions from the mucosa of the upper respiratory organs in abnormal conditions was undertaken in the hope of obtaining information that might be of definite clinical value. We present in this paper determinations of the hydrogen-ion concentration, nitrogen content and viscosity of secretions in the following maladies: allergic rhinitis, colds, sinusitis, allergic rhinitis with polypi, cerebrospinal rhinorrhea and nasal hydrorhea.

METHODS.

The Determination of the Hydrogen-ion Concentration of Nasal Secretion.—The hydrogen-ion concentration is determined by the use of the glass electrode of MacInnes and Dole.[6] The system contains a Leeds and Northrup modified type K potentiometer, a standard cell, storage battery, type F galvanometer, calomel cell, glass electrode, a two micro-farad condenser and a contact key. (Fig. 1.) The circuit is essentially that of Bishop.[7] With this equipment one is able to determine the pH of a solution using only two drops of the unknown. Nasal secretion is collected under oil to prevent loss of carbon dioxide and water. Secretions were permitted to stand six hours and checked against freshly collected secretion. Very little absorption of carbon dioxide took place. The secretion is placed in position (technic of MacInnes and Dole)[1] as rapidly as possible and the chimney filled with paraffin oil. This precaution is necessary to prevent loss of carbon dioxide while the reading is being made. The difference of potential set up between the calomel cell and the glass electrode is carried to the condenser and then discharged through the galvanometer. The voltage on the potentiometer is adjusted until a null point is reached. From this voltage the pH is calculated, using the equation

$$pH_X = pH_S \pm \frac{E_X - E_S}{RT/F}$$

The voltage E_S is that of 0.05 molar acid potassium phthalate, pHs, 3.974. Values of RT/F were taken from the Determination of Hydrogen Ions, Clarke.[8] Readings were made to an accuracy of 0.05 millivolts. Correction for calomel cell to 25° C. was made. Correction to a constant temperature of the secretion was not made as pH per rise of 1° C. is only 0.0035 pH[9]. The system was checked on several occasions with a series of phosphate buffers solution[10] of pH 7.0 to 7.8 with 0.05 intervals. This method, in my hands, has proved much more satisfactory than the capillary quinhydrone method or colorimetric method, using the bicolor standards of Hasting and Sendroy.[11] The superiority of this method is readily understood when one considers the nature and the small volume of the secretion available.

The Determination of Viscosity of Nasal Secretion.—The Hess blood viscosimeter[12] was used to measure the viscosity. The bore of the capillaries of tube A and B, Fig. 2, are identical; then, as one supplies suction simultaneously to the tubes the volume of fluctuation of both liquids will depend upon their degree of viscosity. Tube A is filled to the O mark by suction and the stopcock closed; then the secretion is drawn into tube B in a similar manner to the zero mark. The stopcock is opened and suction applied simultaneously to both tubes until the secretion in tube B has reached point 1, then the reading of the meniscus on tube A is the relative viscosity of the secretion. Nasal secretion was too viscous to make a direct determination, consequently, I used a tenfold dilution. By this method one can secure a relative viscosity. The heterogeneous nature of the secretion, the small amount available and the rapidity with which it loses water are great handicaps in measuring viscosity directly.

Determination of Nitrogen in Nasal Secretion.—Nitrogen is determined by the Koch-McMeekin micro-kjeldahl method.[13] After the addition of the hydrogen peroxide the digestion mixture must be boiled for three minutes to get complete digestion. The technic was first determined on gastric mucin checking with the macro-kjeldahl method and then verified with nasal secretion

whenever sufficient quantities of the secretion were available. The tenfold dilution is usually heterogeneous, containing flakes of tissue like material. This may be corrected by the addition of a drop of caprylic alcohol and shaking vigorously. Determinations were made and proved that the addition of caprylic alcohol did not interfere with the method. Albumen nitrogen was determined by the method of Howe.[14]

Determination of Reducing Substances.—Reducing substances were determined only qualitatively, using Benedict's solution.[15]

The establishment of a so-called normal secretion was the first difficulty to surmount. Measurement of a true normal was out of the question, but we were able to approximate the normal with the use of an irritant. A cone of cantharides plaster, Johnson and Johnson, was placed high in each nares and allowed to remain for twenty minutes (blistering does not occur in this limited time). When the secretion ran from the nostril it was permitted to fall into a tube containing white paraffin oil. At the end of the twenty-minute period the cones were removed and the patient requested to blow the nose, collecting the material in the same tube. Care was exercised to completely cover the secretion. Prior to the treatment with cantharides, a culture was taken from the middle meatus, technic of Linton,[16] for a bacteriological study and smears were made for the cytologic picture.

Table 1 shows the data secured in our normal noses—the mucosa was pale pink, moist and healthy in appearance. The pH in all cases, except N-8, is the same as blood or more alkalin. The nitrogen content varies appreciably and is dependent upon the character of the secretion. The mucin content, calculated from the nitrogen values, varies between 0.77 to 2.41 per cent, with an average of 1.31 per cent. The viscosity of the tenfold dilution has a comparatively narrow range, if case N-9 be omitted. This secretion was very thin and watery. Not enough was obtained for a nitrogen examination.

The data for patients suffering from allergic rhinitis and allergic rhinitis with complications, Table 2 and 3, most closely resemble the normals. The variations of the pH, nitrogen content and viscosity are greater but include the range of the normals. The

allergic secretions are predominantly alkalin, but there are instances (Table 2, patients B. B. and F. G.) whose pH always remained acid. This indicates that there must be two midpoints about which the pH values revolve, the one more alkalin than the normals and the other more acid. This is in accord with the findings of Mittemaier, previously discussed. We note this individual difference among the normals, and again in table 7, patient C. B., in contrast to patients D. W. and P. N., in which the gross appearance of the secretions were identical. The pH in maxillary sinusitis, Table 5, is strongly acid in purulent-fetid secretions, less acid in mucopus secretions and alkalin in the clear mucus secretions. The high alkalinity of the straw colored, serous-like fluid, patients M. C. and A. W., was probably due to a blocked ostium. The preceding determinations, patient A. W., were very acid, as is also true of the later determinations. Thus a single value without the clinical findings could be quite misleading.

A rather interesting correlation between pH and eosinophiles is illustrated, table 3, case H. M. Eosinophiles were present at all determinations except 3/15/33 when the secretion was definitely acid. Protocols B. B. and A. B., table 2, shows the same phenomenon. It would seem that as the pH becomes strongly acid for the individual patient, the eosinophiles are absent. This is in accord with the work of Wenner and Nemours,[17] who found that the removal of ionic calcium from the cytoplasm of the cells of the sinus mucosa produced a local eosinophilia which was augmented by increased blood alkalinity and inhibited by acidosis.

Opie (1904),[18] after an exhaustive study of eosinophilic leucocytes in guinea pigs, reported that these cells are normally present in the intestinal mucosa, in the walls of the trachae, in the bronchi and in the interalveolar walls outside the capillaries. He produced a nutritional eosinophilia by giving the guinea pigs plenty of food and water. Starvation caused a diminution in the number of eosinophiles in the bronchi. Undoubtedly the acidosis of starvation was the cause of this change.

Table 6, patient I. S., was suffering from bilateral maxillary sinusitis. The disease responded quickly to irrigations with normal saline. At first the right antrum had a fetid, greenish-yellow

pus, which became more mucus in character and no odor was detectable, 4/19/33. The following week the washings were clear. A slight increase in acidity occurred as the secretion became more purulent and then decreased as the mucus content increased. The left sinus illustrates an increasing acidity as the secretion became more purulent. However, the exudate contained much more mucus and the pH in consequence was much more alkalin.

I have not been able to correlate the bacterial flora of the nose and sinuses with the pH. The bacteria found are either acid producers or culturally inert. The acids formed, lactic, formic, acetic, butyric, propionic and valerianic, have very little disassociation, consequently the pH would not be appreciably changed. Where carbon dioxide or hydrogen sulphide are formed the pH is no doubt affected. One striking feature is observed in tables 5 and 6. All the patients with pH 6.5 or below always gave a negative culture, although many bacteria were present in the antral smear. Undoubtedly, the low acidity has been unfavorable to the growth of the pathogenic bacteria. It is possible that a toxin produced together with the low pH is responsible for the death of the bacteria. Patient C. O., pH 5.84, table 5, gave a positive culture of hemolytic staphylococcus albus. This culture was taken from the middle meatus after collection of the crusty exudate; thus, it was taken from the surface of the living tissue where the pH would more nearly approximate the pH of the blood. If it had been possible to secure a culture from the tissue of the maxillary sinuses already mentioned a growth might have been obtained.

The variation in nitrogen content is tremendous. Table 8 shows the minimum and maximum values for each disease. Excepting the secretion from a cerebrospinal rhinorrhea, which is not an exudate of the mucosa of the nares and accessory sinuses, the lowest nitrogen is found in allergic rhinitis. This represents nonprotein nitrogen and mucin. Allergic rhinitis accompanied with polypoid tissue produces a secretion which contains much more nitrogen. Albumen was present when the nitrogen values were about 1200 mg. per cent. The amber colored secretions contained more albumen. Dehydration of the material in the nose must play an important rôle in these instances. In sinusitis the nitrogen

ran very high; the more purulent the secretion the higher the nitrogen content. As the exudate changes from predominately pus to predominately mucopus, the nitrogen values decrease, illustrated in table 6. A survey of the tables shows that nitrogen is always high in inflammatory suppurations. The thin, watery secretion of colds is very low in nitrogen, whereas the opalescent and yellow exudates from the later stages of the disease contain much more nitrogen.

Nitrogen content is dependent upon the nature of the secretion. The presence of albumen or leucocytic infiltration rapidly increases the amount of nitrogen. Dehydration, due to long standing in the nose, is also an important factor. A simple allergic rhinitis does not contain albumen. A study of the amino acid nitrogen, ammonia nitrogen, creatinin and albumen of the exudate of the mucous membrane of the upper respiratory organs would be interesting and might bear a relationship to the bacterial flora. Similar determinations should be made on the blood simultaneously.

The viscosity of the tenfold dilution of the secretion is dependent upon the nature of its content. A purulent fetid secretion is less viscous than a mucopus secretion, table 7. The viscosity of allergic rhinitis most closely resembles that of the normals, table 8. The range of the former is greater and includes the variation of the latter. The exudate of allergic rhinitis accompanied with much polypose tissue containing albumen is much more viscous than the normals, table 8. As the improvement in the clinical picture takes place in this disease, the viscosity more nearly approximates the normals, table 4, patients S. K. and E. D. Both patients reported no more nasal secretion and both are still well. Viscosity cannot be correlated with nitrogen content or the hydrogen-ion concentration. Long standing in the nose with subsequent dehydration is also an important factor.

A cerebrospinal rhinorrhea can very easily be detected by physicochemical tests. Dr. W. H. Johnston, Santa Barbara, Cal., sent me a secretion he had obtained from a patient diagnosed as cerebrospinal rhinorrhea. An examination showed a direct viscosity of 1.1 at room temperature, an extremely low nitrogen,

19.6 mg. per cent, and the presence of a reducing substance, probably glucose. Only an occasional cell was found in the microscopic study. These values are identical with the values of cerebrospinal fluid. The second patient appeared a few days ago in our outpatient clinic. She gave a history of yawning largely after a bath and shortly thereafter tasting something in her mouth that she thought was blood. She expectorated and found only a clear secretion. When she sat up the secretion began to drip from her nose. She was able to collect about 10 cc. in twenty-five minutes. Examination of the clear fluid showed a direct viscosity of 1.1 at room temperature, 20.8 mg. per cent nitrogen and the presence of a reducing substance. These values are again like those of cerebrospinal fluid. The secretion did not stiffen a handkerchief. This would also indicate that there is an absence of protein. The only case that I have that approximates these is of a patient that was sent to the hospital as a suspected cerebrospinal rhinorrhea. The direct viscosity was 7.2 to 15.6, nitrogen 88 mg. per cent and no reduction with Benedict's solution could be observed. This, together with the clinical findings, indicated a nasal hydrorrhea. At no time have I been able to get a positive sugar from any type of nasal secretion unless it was contaminated with blood. The hydrosylate of pus did not show the presence of a reducing substance. Israel and Nicholas[4] reported identical results. However, the hydrosylate of mucopus and the mucus secretions always showed the presence of a reducing substance, which no doubt originates from the carbohydrate fraction of mucin. Hydrolyzed mucin always has considerable reducing power.

SUMMARY.

A physical-chemical study of secretions from normal mucous membrane and from varying diseases of the nose and accessory sinuses was made.

Cantharides plaster was used to obtain a secretion to establish normal values.

The hydrogen-ion concentration for each disease can be placed in two groups, the one more acid than the normals, and the other more alkalin, with the one of the two groups predominating.

The degree of acidity or alkalinity is dependent upon the purulency of the secretion, the mucus content and a probable constitutional factor.

The fluid from a cerebrospinal rhinorrhea is readily differentiated from the secretion of the mucous membrane of the nose by a chemical examination.

Nitrogen content and viscosity are both dependent upon the character of the secretion.

TABLE I. NASAL SECRETION OBTAINED FROM NORMAL NOSES.

Patient	Viscosity Dil. 1:10	pH	Nitrogen mg. %	Character	Cytology	Bacteriology
N- 1	1.43	7.73	220	Faint amber, tinged with blood	Neutro., epith. cells	No growth
N- 2	1.52	7.88	184	Clear	Epith. cells	No growth
N- 3	1.53	7.49	215	Clear, slightly opalescent	Neutro., epith. cells	Staph. albus
N- 4	1.33	7.52	385	Opalescent	Neutro., epith. cells	Staph. albus
N- 5	1.37	7.54	202	Clear, opalescent, yellow	Eosin., neutro., epith. cells	No growth
N- 6	1.42	7.40	124	Clear, white flakes	Epith. cells	Staph. albus
N- 7	1.42	7.40	150	Clear, white flakes	Few neutro., epith. cells	Hemo-staph. diphtheroids
N- 8	1.56	7.19	194	Clear, yellow clumps	Neutro., epith. cells	Hemo-staph. diphtheroids
N- 9	1.10	7.57	.....	Clear	Neutro., epith. cells	No growth
N-10	.....	7.72	.....	Clear	Neutro., epith. cells	Many hemo-staph. albus

TABLE II. ALLERGIC RHINITIS.

Patient	Date	Viscosity Dil. 1:10	pH	Nitrogen mg. %	Character	Cytology	Bacteriology
F. R.	1/18/33	1.56	7.44	226	Sl. opalescent	Neutro., epith. cells	No growth
"	1/27/33	1.40	7.66	163	Sl. opalescent	Neutro., epith. cells	No growth
"	2/10/33	.....	7.49	426	Opalesc., yellow clumps	Neutro.	Diphtheroids m. catarrhalis
"	2/17/33	1.55	7.44	496	Opalesc., yellow clumps	Neutro., eosin., epith. cells, bacteria.	
"	2/24/33	.....	7.58	.....	Clear	Bacteria, epith. cells	
F. G.	2/28/33	1.75	7.05	263	Clear, tenacious	Neutro., eosin., epith. cells	No growth
"	3/16/33	1.62	7.32	267	Clear, few yellow clumps	Neutro., eosin., incapsulated diplococci	
M. J.	2/24/33	1.46	7.48	301	Opalescent	Neutro., eosin., epith.	Hemo-staph. albus diph-theroids m. catarrhalis
B. B.	2/ 4/33	1.12	7.36	66	Opalescent	Neutro., eosin., epith. cells	Staph. albus
"	2/13/33	.....	7.11	.....	Clear	Neutro., eosin., epith. cells	No growth
"	2/20/33	1.33	7.31	150	Clear, whitish specks	Neutro., eosin., epith. cells	Hemo-staph. albus
"	2/24/33	.....	6.74	.....	Clear	Neutro., epith. cells	No growth
A. B.	1/21/33	1.34	7.23	108	Clear, whitish specks	Neutro., epith. cells	
"	2/24/33	1.22	7.70	88	Clear	Neutro., eosin., epith. cells	

Some cases of allergic rhinitis are always acid, namely, B.B. and F.G. eosinophiles were not present in the secretions from B.B. and A.B. when the pH was definitely acid for the individual.

TABLE III. ALLERGIC RHINITIS.

Patient	Date	Viscosity Dil. 1:10	pH	Nitrogen mg. %	Character	Cytology	Bacteriology
E. H.	9/14/32	1.77	7.63	525	Clear, yellow clumps	Eosin., neutro., bact.	
"	9/16/32	1.80	7.53	142	Clear, profuse	Eosin., neutro., bact.	
"	9/17/32	1.70	7.55	223	Opalescent	Eosin., neutro., bact.	
"	9/18/32	1.55	7.95	108	Clear	Eosin., neutro., bact.	No growth
"	9/20/32	1.37	7.51	87	Opalescent	Eosin., neutro., bact.	
"	9/22/32	1.61	7.62	169	Opalescent	Eosin., neutro., bact., epith. cells	
"	9/24/32	1.35	7.42	90	Clear	Eosin.	
"	9/26/32	1.24	8.11	156	Clear, profuse	Eosin., neutro., epith. cells	Hemo-staph. albus
"	9/27/32	1.23	7.58	131	Clear	Eosin., neutro., epith. cells	
"	9/28/32	No secretion				Eosin., neutro., epith. cells, bact.	
H. M.	9/24/32	1.37	7.46	229	Opalesc., yellow clumps	Eosin., neutro.	
"	9/30/32	1.39	7.81	108	Clear, few pale flakes	Eosin., epith. cells	
"	10/ 3/32	1.57	7.36	102	Clear, few pale flakes	Eosin., neutro.	No growth
"	10/ 7/32	1.47	7.86	156	Clear, few yellow clumps	Eosin., neutro.	No growth
"	10/ 8/32	1.78	7.87	120	Opalescent	Eosin., neutro.	No growth
"	10/10/32	1.48	7.79	138	Clear, few pale yellow clumps	Eosin., epith. cells	No growth
"	10/11/32	1.39	7.55	101	Clear, very few pale flakes	Eosin., neutro., epith. cells	No growth
"	10/12/32	1.47	7.52	62	Clear	Eosin., neutro., epith. cells	No growth
"	10/13/32	1.48	7.56	127	Clear, few white specks		
"	1/27/33	1.32	7.58	94	Clear, very few white specks	Eosin., neutro., epith. cells	No growth
"	2/15/33	1.13	7.13	97	Clear, very few white specks	Neutro., epith. cells, bact.	Hemo-staph. albus
"	2/24/33	1.36	7.86	136	Clear, more tenacious	Eosin., neutro., epith. cells, bact.	No growth

E. H. had a seasonal allergy; H. M. had a food allergy. Highest pH values were obtained when the secretion was most profuse. Eosinophiles disappeared when the secretion became definitely acid, patient H. M., 2/15/33.

TABLE IV. ALLERGIC RHINITIS WITH POLYPI.

Patient	Date	Viscosity Dil. 1:10	pH	Nitrogen mg. %	Character	Cytology	Bacteriology
R. W.	7/19/32	4.76	7.56	485	Pale yellow, tenacious	Neutro., bacteria	
"	7/22/32	4.20	.....	517	Pale yellow		
E. W.	10/ 4/32	3.57	7.11	1847	Amber yellow	Neutro., epith. cells, bact.	Hemo-staph. albus
S. K.	6/24/32	4.64	7.40	750	Pale yellow		
"	7/ 8/32	.....	7.59	...	Deep amber, gristle like		
"	9/30/32	.....	7.42	1299	Amber yellow	Eosin., neutro., epith. cells	Hemo-staph. albus
"	1/27/33	3.02	7.07	735	Pale yellow and whitish	Eosin., neutro., epith. cells, bacteria	No growth
"	2/ 3/33	.....	7.44	.....	Very pale yellow	Eosin., neutro., epith. cells	No growth
"	2/23/33	2.70	7.49	946	Clear, pale yel. clumps	Eosin., neutro., epith. cells, bacteria	Staph. albus hemo-steph.
"	3/24/33	Reported no more nasal discharge.					
E. D.	9/15/32	.....	6.99	.....	Pale yellow	Eosin., neutro., bacteria	No growth
"	10/ 7/32	too thick	7.05	.....	Medium yellow clumps	Eosin, neutro., epith. cells, bacteria	No growth
"	10/ 8/32	.....	7.13	.....	Clear and opalescent	Eosin., neutro., epith. cells, bacteria	Hemo-staph.
"	10/10/32	4.2	7.19	2264	Amber and pale yellow	Eosin., neutro., epith. cells	Hemo-staph. albus, m. catarrhalis
"	10/11/32	2.9	7.27	766	White with yellow flakes	Eosin., neutro., epith. cells, bacteria	Hemo-staph. albus, m. catarrhalis diphtheroids
"	10/12/32	2.30	7.07	1211	Deep yellow	Eosin., neutro., bacteria	No growth
"	10/13/32	2.30	6.93	1258	Yellow and amber yel.		
"	12/ 9/32	.....	6.95	.....	Clear		

Viscosity decreases approaching the normal as clinic improvement takes place. High nitrogen due to the presence of albumen.

TABLE V. SINUSITIS.

Patient	Date	Viscosity Dil. 1:10	pH	Nitrogen mg. %	Character	Cytology	Bacteriology
M. C.		.....	8.13	.....	Serous-like gel.	Bacteria, neutro., epith. cells	
H. P.	9/15/32	.....	7.40	.....	Clear, tinged with blood		Staph. aureus
"	9/19/32	1.49	7.76	124	Clear, sl. opalescent		Staph. albus
M. B.		3.89	7.14	883	Opalesc., tenacious, odor		
C. S.		.....	7.41	327	Clear, white flakes	Neutro., eosin., epith. cells	Hemo-staph. albus, diphtheroid
A. H.		1.77	6.82	530	Opaque, whitish	Neutro., eosin., epith. cells	Hemo-staph. aureus
W. N.	1/26/33	3.71	7.59	684	White, tinged with blood	Neutro., eosin., epith. cells, fibrin.	No growth
"	3/16/33	.....	7.19	125	Clear	Neutro., eosin., epith. cells	No growth
M. T.		.....	6.31	2202	Yellow clumps	Neutro., eosin., fibrin, monocytes, lymphocytes	No growth
C. O.		.....	5.96	3306	Crusty	Neutro., epith. cells	Staph. albus
G. S.	2/13/33	.....	6.69	1781	Fetid yellow pus	Neutro., epith. cells, bact.	B. Proteus
"	2/21/33	2.01	7.06	1601	Fetid yellow pus	Same as above.	B. Coli
S. M.	3/22/33	1.42	6.53	1067	Fetid yellow pus		
A. W.	3/10/33	.....	6.06	1849	Yellow pus	Monocytes, lymphocytes, neutro., eosin.	No growth
"		.....	6.77*	2175*	Yellow, pink with blood	Monocytes, lymphocytes, neutro., eosin., bacteria	No growth
"	3/16/33	.....	6.13*	1474	Yellow pus	Neutro., eosin., bacteria, monocytes, lymphocytes	No growth
"	3/23/33		8.04*	.....	Serous-like gel.	Bacteria, neutro., epith.	No growth
"	3/28/33	.....	6.29*	1880	Yellow pus		No growth
W. D.	3/24/33	.....	5.86*	837	White pus	Neutro., epith. cells, eosin., lymphocytes, bacteria	No growth
A. G.	4/12/33	1.4	7.15		Straw-colored fluid with lumps of mucopus	Neutro., eosin.	Hemo-strep., hemo-staph.

Purulent secretions are acid in character. Nitrogen values are dependent upon the character of the secretion. High alkalinity, pH 8.13 and pH 8.04, are probably due to a blocked ostium.

* Exudate from the antrum.

TABLE VI. PATIENT I. S. BILATERAL MAXILLARY SINUSITIS.

Date	Viscosity Dil. 1:10	pH	Nitrogen mg. %	Character of Secretion	Bacteriology
Right Maxillary Sinus					
3/22/33	1.2	6.14	1236	Fetid, yellow pus, tinged with blood	No growth
3/29/33	.....	6.10	1494	Fetid, yellow pus	No growth
4/ 5/33	1.4	.....	1774	Fetid, greenish-yellow pus	No growth
4/12/33	1.4	6.32	1191	Fetid, yellow pus with some mucus	No growth
4/19/33	.....	.....	.....	Too thick to secure	
4/26/33	Washings Clear				
Left Maxillary Sinus					
3/29/33	.....	7.24	1390	Yellow pus, some mucus, tinged with blood	H. influenzae
4/ 5/33	1.5	.....	1021	Whitish yellow pus, mucus	No growth
4/12/33	1.5	7.00	667	White pus, more mucus than above	H. influenzae, m. catarrhalis
4/19/33	1.5	6.86	676	Pale greenish-yellow pus, more mucus than 4/12/33	Many gram negative diplococci and m. catarrhalis
4/26/33	.....	.....	.....	Too thick to secure	
5/ 2/33	Washings Clear				

pH increases as the secretion becomes more mucoid and decreases as the exudate becomes more purulent. Nitrogn content increases as the secretion changes from a yellow pus to a greenish-yellow pus, and decreases with an increasing mucus content.

TABLE VII. COLDS.

Patient	Date	Viscosity Dil. 1:10	pH	Nitrogen mg. %	Character	Cytology	Bacteriology
C. W.	9/24/32	.....	7.39	.....	Pale yellow	Eosin., neutro.	
"	9/26/32	1.86	7.69	303	Pale yellow	Neutro.	No growth
"	9/27/32	2.15	7.47	523	Pale yellow	Eosin., neutro., bact.	
"	9/28/32	1.95	7.45	745	Deep yellow	Eosin., neutro., bact.	No growth
"	9/29/32	2.48	7.66	536	Opalescent, tinged yel.	Eosin., neutro.	
"	9/30/32	2.27	7.15	619	Pale yellow	Eosin., neutro.	
D. W.	10/ 1/32	.....	8.04	.....	Clear, few yellow specks	Neutro., epith. cells	Diphtheroids hemo-staph. albus
P. N.	9/24/32	1.10	8.04	.....	Clear	Epith. cells	
"	12/ 6/32	1.64	7.83	125	Clear, white flakes	Eosin., neutro., epith. cells	Diphtheroids
W. W.	12/ 8/32	2.02	7.35	377	Opalescent, white	Neutro., epith. cells, bacteria, yeast	Hemo-staph. albus
"	12/14/32	2.06	7.64	149	Clear	Neutro., epith. cells	No growth
"	1/16/33	1.58	7.69	194	White, yellow clumps	Neutro., epith. cells	No growth
"	1/23/33	1.32	6.78	903	Opalescent, yel. flakes	Eosin., neutro., epith. cells	
"	2/28/33	2.37	7.39	509	Opalescent, tenacious	Neutro., epith. cells	No growth
L. L.	2/ 7/33	.....	7.77	.....	Clear, slightly opalescent	Neutro., epith. cells	Hemo-staph. albus
C. B.	2/27/33	.....	6.59	.....	Clear	Epith. cells	No growth
"	3/17/33	.....	7.43	.....	Clear, slightly opalescent	Neutro., epith. cells	No growth

A striking difference is shown in the pH of the clear secretions from patients P. N. and D. W., in contrast with that of patient C. B. All were suffering from the initial stages of a cold.

TABLE VIII. RANGE OF NITROGEN CONTENT AND VISCOSITY OF NASAL SECRETIONS.

	Nitrogen mg. %	Viscosity 10-fold dilution
Normals	123 — 385	1.33 — 1.56
Allergic Rhinitis	62 — 585	1.10 — 1.80
Allergic Rhinitis with Polypi	485 — 2264	4.76 — 2.30
Sinusitis	124 — 3506	1.22 — 3.89
Colds	125 — 903	1.32 — 2.48

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LXXXII.

CONGENITAL ANOMALIES OF THE ESOPHAGUS, WITH SPECIAL REFERENCE TO THE CONGENITALLY SHORT ESOPHAGUS WITH A PORTION OF STOMACH ABOVE THE DIAPHRAGM.*

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PHILADELPHIA.

In considering congenital anomalies of the esophagus one invariably refers to the common form, which is characterized by atresia associated with an esophagotracheal fistula between the lower esophageal segment below, and the posterior wall of the trachea slightly above the bifurcation, more rarely the right bronchus, above. This form is the most serious and is invariably fatal. Of the nonatresic varieties, Findlay and Kelly[1] have recently described a form characterized by congenital shortening of the esophagus with the occurrence of a thoracic stomach. Our interest in this group was stimulated by the findings in a patient admitted for removal of a bolus of food that had lodged in the esophagus, and the subsequent study of several cases that had originally been diagnosed as peptic ulcer of the esophagus. In Findlay and Kelly's cases, nine in number, it was at first believed that the condition was a diaphragmatic hernia of the stomach.[2] By careful roentgen study and by esophagoscopy they later demonstrated that the tube extending between the stenosis and the diaphragm was greatly dilated and was lined by gastric mucosa.

The scarcity of the literature bearing on this subject certainly should not indicate that the condition is rare. On the basis of our discovery of nine cases in the past three years it must be relatively common. Cases are probably overlooked because of the lack of an adequate roentgen study and also failure of the esophago-

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scopist to carefully examine the food passages distal to the stenosis. In four of our cases a diagnosis of peptic ulcer of the esophagus at the hiatus esophageus had previously been made. Ulceration was present, but this occurred at a point considerably higher than the hiatal level. The stenosis was explained as resulting from healing of a part of the ulcer. That portion of the food passages beyond the point of ulceration was not examined by the esophagoscopist, consequently no opinion regarding its character was expressed. It is entirely probable that many of these cases are diagnosed and treated as stricture, peptic ulcer, hernia of the diaphragm, deformity of the esophagus, or cardiospasm, and that others will never come to light at all.

In an analysis of our series of nine cases certain previously unrecorded observations were made that are noteworthy. It was interesting to note that two of the patients were brother and sister. Age and sex: In Findlay and Kelly's[1] series, which consisted of seven boys and two girls, the ages varied from four weeks to nine years and ten months. Our group included one girl and three boys, varying from six and one-half to nine and one-half years of age, and five women, varying from 48 to 64 years.

Symptoms.—In view of the fact that but one group of cases has been reported, namely, that of Findlay and Kelly, no classical symptom group has been suggested. In their series, vomiting or regurgitation of food with disturbances in growth and nutrition were the outstanding symptoms, being present in all cases. In seven, regurgitation was present from birth. Although dysphagia was never mentioned by the parents, it is apparent that this symptom was invariably present. From the standpoint of symptoms our cases could well be divided into two groups. In one, consisting of three children, the outstanding symptoms were dysphagia, regurgitation of food and disturbances in nutrition and growth. The other group, consisting of one child, aged 6½ years, and five adults, the symptoms were not only those of disturbances in swallowing with lodgment and regurgitation of food, but in addition distress, particularly after eating. This varied from flatulence and indigestion to severe epigastric pain.

Dysphagia.—In the four children and in one adult the difficulty in swallowing was present since birth. Solid foods could not be swallowed, and in one case only liquids could be taken without difficulty. In the remaining four adults, difficulty in swallowing was present for "many years" to twenty years. Several found it necessary to take large quantities of fluid when eating, to aid in swallowing. Regurgitation was not uncommon and always occurred during meal time. Lodgment of food was a frequent symptom; five of the patients were referred to the Bronchoscopic Clinic because of aphagia resulting from obstruction by food (Fig. 3).

Weight Loss.—This was particularly noticeable in the children, all of whom were underweight and poorly developed. In the case of the child, aged 8 years, the dysphagia with emaciation was so marked that the patient was sent to a surgeon for gastrostomy, a diagnosis of cicatricial stenosis of the esophagus having been made, although there was no history of injury to the esophagus. In the adults disturbances in nutrition were not marked—in fact, three of the patients were well nourished.

Pain.—Distress varying from flatulence and indigestion to severe epigastric pain was present in the five adults and in one child. This occurred very shortly after the taking of food and in three cases it was severe. It was usually referred to the epigastrium and behind the lower half of the sternum; in two instances it was also referred to the back.[3] In five of the six cases, sodium bicarbonate was frequently taken for relief of pain. In two of these, acid foods produced severe distress. It was interesting to note that one patient experienced severe pain on assuming a recumbent position—in fact, she could sleep only in a semi-sitting posture. At esophagoscopy it was found that there was extensive superficial ulceration at the gastro-esophageal junction. The pain was very probably due to contact of gastric juice with the area of ulceration which occurred when she assumed a recumbent posture.

Roentgen Findings.—The essential points in the roentgen ray diagnosis of congenital shortening of the esophagus are, first, a portion of the cardiac end of the stomach must be shown to stay above the level of the diaphragm; second, the esophagus must

be shown to be too short to reach as low as the level of the diaphragm. As to the first point, the only characteristic sign is the presence of longitudinal rugae markings in that portion of the tube just above the diaphragm.

When this point is established, then change of posture will not cause that portion of the stomach above the diaphragm to go either lower or higher, and there will be no variation in relations on repeated studies.

If it is to be viewed at all in the light of a diaphragmatic hernia or hernia of the stomach through the diaphragm, then the term "congenital fixed" or "congenital irreducible" should qualify the term "hernia."

There are other contributory signs, most important of which is that the portion of the stomach above the diaphragm will only show its true diameter or capacity when that portion below the diaphragm is well filled (Fig. 1), and then only to the best advantage when the patient is in the right-oblique-prone posture, previously described, in which position the hiatus is on a higher level than the upper end of the esophagus, and gravity tends to keep the barium mixture from going into the abdominal portion of the stomach.[4] In fact, in one of our cases we did not succeed in getting diagnostic evidence until the patient was placed in this posture (Fig. 2). Having demonstrated a lumen wider than the normal esophagus above the level of the hiatus where there is no evidence of obstruction, either organic or spasmodic, at the diaphragm, the evidence is strongly contributory.

Roentgenographically all of our cases have shown some degree of narrowing where the esophagus joins the stomach. This appearance of narrowing does not differ greatly from that seen in strictures following injury except that in the vast majority of instances the narrowing due to cicatricial tissue formation following injury is much more marked when we first see it. The portion of the esophagus below the stricture is not dilated, the history is that the difficulty in swallowing has been progressive, and there is apt to be more or less emaciation, while in the congenital shortening cases the difficulty to swallow a large bolus of solid food has always been present. The narrowing at times

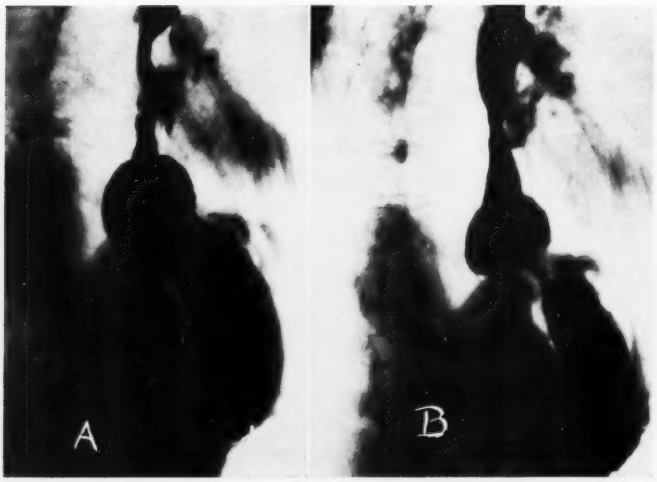

Fig. 1. Roentgenograms made in the case of a woman, aged 53 years, who has complained of dysphagia and indigestion for 18 years. These indicate the position of the cardiac end of the stomach above the level of the diaphragm. In A, the maximum filling of that portion of stomach above the diaphragm is observed when the subdiaphragmatic portion is filled. In B, rugae are noted in that part of the stomach that passed through the diaphragm. Attention is also directed to the extreme width of the opening through the diaphragm in both illustrations.

causes so little inconvenience that the diagnosis is overlooked entirely or is only by chance brought to light in adult life. On the other hand, the lumen has been rather small in a few of our cases; in one the condition was brought to light only after complete obstruction by the esophagus by a bolus of food (Fig. 3). The narrowing has been rather uniform as to location, being at about the level of the seventh or eighth dorsal vertebra.

As reported by Findlay and Kelly,[1] we found no collection of air or gas in the stomach, either above or below the diaphragm. In none did we find any appreciable evidence of delay of the barium mixture at the diaphragm level, and in a few of them the opening in the diaphragm seemed to be appreciably larger than

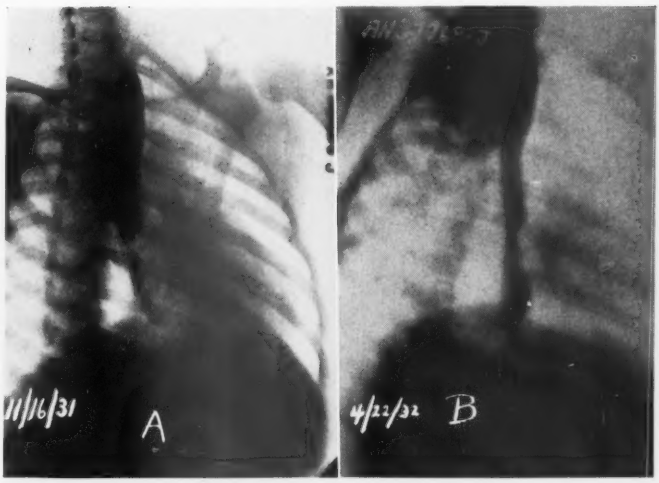

Fig. 2. Roentgenograms exhibiting the importance of proper filling to demonstrate the dilated portion above the diaphragm. In A, with the patient in the erect posture, the short narrow lumen is observed at the junction of the esophagus with the stomach. In B, with the patient in the right-oblique-prone posture filling of the stomach below the narrowing is noted.

normal (Fig. 4). In one case definite evidence of the condition was seen roentgenoscopically, but satisfactory roentgenograms were not secured—in fact, it was not realized that the case was one of this type until we later noted the significance of the report.

As previously noted, two of our group were of one family, brother and sister, but two other sisters and three other brothers showed no abnormality.

In all of our cases it was evident that the esophagus proximal to the narrowing was not long enough to reach the diaphragm in any posture or state of filling with any consistency of barium mixture. A few of them show evidence of dilatation proximal to the narrowing (Fig. 5).

Roentgenoscopic views in anteroposterior and lateral projections, with the patient both standing and lying down, have shown an entire absence of tortuosity of the esophagus.

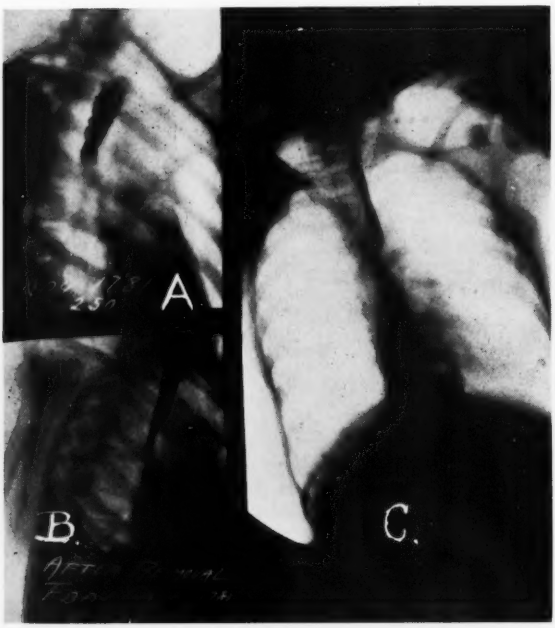

Fig. 3. Roentgenograms made in the case of a girl, aged nine and a half years, who was brought to the Bronchoscopic Clinic because of inability to swallow after choking on a bolus of meat. There was a history of dysphagia since birth. In A, there is noted complete obstruction of the esophagus produced by a bolus of food. In B, with the patient in the right-oblique-prone posture there is observed complete filling below the narrowed lumen. In C, with the patient in the recumbent position, the spine interferes radiographically. This is the sister of the patient in Fig. 2.

Esophagoscopic Findings.—At esophagoscopy there was invariably found dilatation of the upper thoracic esophagus with a chronic esophagitis. The esophagus was shorter than normal as shown by measurement and by anatomic localization. Its lower end was approximately opposite the fourth rib, along the left border of the sternum. Unlike the normal hiatus, the narrowing found at the lower end of the esophagus offered marked resistance to the tip of the esophagoscope, suggesting an organic

Fig. 4. On this roentgenogram is observed narrowing at the junction of the esophagus and cardiac end of the stomach well above the diaphragm. Typical rugae shadows are noted immediately above the opening in the diaphragm. This patient weighs 250 lbs. She has had "stomach trouble" for many years.

stenosis. In two cases there was a weblike stenosis at this point; in a third, the narrowing was very abrupt with a small lumen more or less concentrically placed and about 5 mm. in diameter. In five cases the stenosis was funnel-like in character, extending over a distance of 2 to 4 cm. In the remaining case there were several points of narrowing, suggesting several strictures with a large, funnel-like narrowing above.

In four cases ulceration was present, varying from a small area at the point of stenosis to extensive ulceration involving not only the funnel-like narrowing but the stenosis itself. The ulceration appeared superficial with an inflammatory edge and was covered by a thin grayish exudate. Granulations, when present, were usually flat. Immediately on traversing the stenosis the esopha-

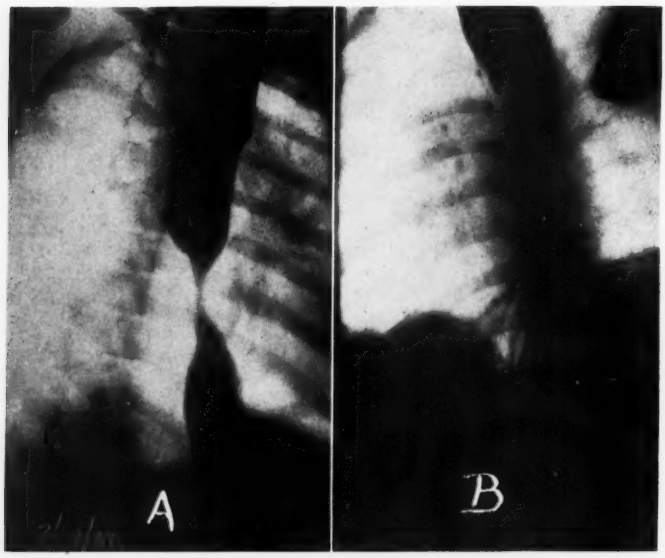

Fig. 5. Roentgenograms of the esophagus made in the case of a patient, aged 8 years, who, because of marked dysphagia and emaciation required gastrostomy. There is a history of dysphagia since the patient was three weeks old. In A, is noted marked narrowing of the lumen at the junction of the esophagus and stomach. There is marked dilatation of the esophagus proximal to the narrowing. In B are observed details of rugae in the stomach above the diaphragm.

goscope entered into what appeared to be the stomach, although anatomically the tip of the esophagoscope was still above the level of the diaphragm. This fact may be readily overlooked unless one routinely makes accurate comparative measurements. The stomach mucosa appeared normal. In no case was it possible esophagoscopically to observe the narrowing in the stomach which would correspond to the level of the diaphragm, normally the hiatal level. The patient with a gastrostomy fistula was examined by retrograde methods on a number of occasions to determine this point. Viewed through the retrograde gastroscope one could not differentiate between the findings in this case and those in the

normal individual except that the gastro-esophageal junction offered resistance to the gastroscope which normally would not be met with. From an esophagoscopic standpoint, one gained the impression that as soon as the stenosis was passed one entered the stomach.

In all of the cases tests were made with litmus paper after the stenosis was passed, to ascertain the reaction of the fluids. It was invariably acid. This in itself is of little importance, for it is a known fact that regurgitation of gastric content is not uncommon when performing an esophagoscopy on a patient without anesthesia. To corroborate the esophagoscopic impression that gastric mucosa was observed at a point proximal to the level of the diaphragm, specimens of tissue were removed in seven cases. In all of these the histologic report indicated the presence of normal gastric mucosa. In several, the histologic appearances of tissue removed from the area of ulceration suggested that the lesion was a peptic ulcer.

One is always confronted with the question whether these are not cases of acquired stenosis, particularly if the patient first manifested symptoms late in life. Although no history of swallowing caustics or other injury to the esophagus could be elicited in the cases reported, it is quite possible that such an accident could occur during the first few years of life and would not be remembered. Irrespective of these doubts, one does not find a similar combination of anatomic changes such as were observed in these cases that could possibly have resulted from any form of injury.

Treatment.—In the cases of web stenosis, dilatation was readily carried out, as the web itself offered no resistance to the tip of the esophagoscope and bougie. In the nonulcerated cases esophagoscopic bouginage was very promptly followed by improvement. This was particularly evident in the children. The patients with ulceration at the junction of the esophagus and stomach presented a difficult problem in treatment. Pain was the most prominent symptom. Various combinations of alkalies in solution and bismuth subnitrate, dry on the tongue, afforded temporary relief Topical applications of silver nitrate, 10 per cent, to the ulcerations seemed to be helpful. In one case dilatation of the stenosis

was followed by complete relief of pain, although ulceration still is present.

Dietetic treatment along the lines indicated in ulcer of the stomach should be followed. Esophagoscopic dilatation is indicated until swallowing of properly masticated foods can be normally performed.

Summary.—Nine cases of congenital shortening of the esophagus with stenosis and the presence of a portion of stomach in the thoracic cavity are reported. The group consisted of four children and five adults. Two children were of one family, brother and sister. Two groups of symptoms were noted, one consisting of those resulting from esophageal obstruction and malnutrition; in the other, symptoms due to ulceration were predominant. The essential points in the roentgen diagnosis are presented and discussed. The esophagoscopic findings are noted. The condition is believed to be more common than medical literature would indicate.

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LXXXIII.

PHYLOGENIC DEVELOPMENT OF THE COCHLEA.*

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Renewed interest in study of the physiology of hearing has resulted from the report by Wever and Bray, in 1930, on "The Nature of Acoustic Responses." The work since then has been concentrated on interpretation of physiologic experiments on the auditory mechanism. To formulate any theory of hearing, it is important to consider not only the anatomic and physiologic evidence but also the evolutionary development of the cochlea. In tracing the growth of the cochlea from the lowest to the highest of the vertebrates, definite evidence can be found of the way this organ may respond to sound.

The subject will be developed in the following order:

1. Organ of hearing in the fish and its phylogenic importance.
2. The development of the organ of hearing in the amphibian, reptile and bird.
3. Organ of hearing in the lowest of mammals and its relationship to higher forms.
4. The neuro-anatomic and physical evidence in the mammalian cochlea of its double development.
5. Conclusion.

I. The lowest order of vertebrates are the leptocardii (acrania). The amphioxus lanceolatus, a member of this order, shows no evidence of any auditory mechanism. In the next order, cyclostomi (marsipobranchi), the auditory organ consists of one or two semicircular canals united in a common section called the saccus communis. Fig. 1—A drawing of the auditory organ in the hagfish (myxine glutinosa). From this simple saccus communis develop the utricle, saccule, lagena and eventually the

*Read at the American Otological Society, May 9, 1933, at Washington, D. C.

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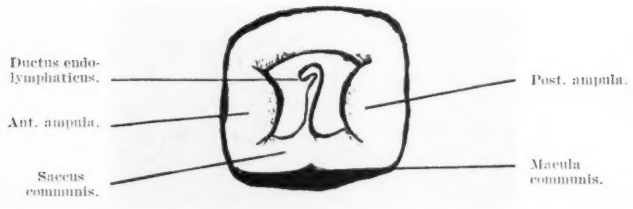

Fig. 1. Hagfish.

Membranous labyrinth of the hagfish consisting of semicircular canal with two ampula and has a saccus communis.

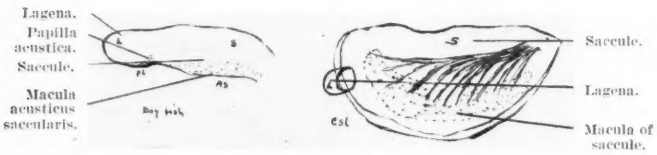

Fig. 2. Dogfish—Skate.

Showing relationship of the lagena to the saccule and the neuro-epithelium of the lagena to the saccule.

Fig. 3. Codfish.

Showing relationship of lagena to saccula nerve supply to lagena and to saccule. Also showing canal connecting saccule and lagena.

mammalian cochlea. The saccus communis has a macula (neuro-epithelial sense organ) and contains an otolithic mass which stimulates the macula by its vibrations. All vertebrates above this order have three semicircular canals, utricle, saccule and lagena (cochlea).

The true fishes, cartilaginous and bony, have a saccule and lagena. The lagena, or primitive cochlea, is a small knoblike prolongation from the saccule and opens freely into the cavity of the saccule by a canal (canal sacculo-lagena). The lagena cavity is filled with endolymph and an otolithic mass. There is a definite neuro-epithelial sense organ present (papilla acustica lagena). This neuro-epithelial organ is made up of hair sense cells of a character similar to that of the macula acustica of the saccule. Fig. 2, dogfish (Sylidae) and skate (Rajides), shows the relationship of the lagena to the saccule and that of the papilla acustica lagena to the macula acustica sacculari*s*. As the vertebrate develops into a higher order of fish the papilla acustica of the lagena increases in importance and the size of the lagena also increases. Fig. 3—Codfish (Gadidae). Fig. 4—The carp (Cyprinidae) family shows the highest development of the lagena in fishes. The nerve supply to papilla acustica of the lagena is as great as that to the macula of the saccule. The lagena has become greatly enlarged at the expense of the saccule.

Warner, in an article, "The Sensitivity of Fishes to Sound and other Mechanical Stimulation," gives the following summary in regard to the response of fish to sound:

"There are wide specific differences in sensitivity to sound. Some species may be deaf. There are also differences in the range of effective frequencies. Some fishes can apparently discriminate tones differing considerably in pitch. But generally speaking, the hearing of fishes is certainly inferior to that of air-living vertebrates. Sounds produced under water are more effective stimuli than those produced in the air above the water. Fishes appear to be far more sensitive to the lower frequencies, and most sensitive to frequencies below those falling under the term sound. It is probable that the ear is the receptor for tones of several hundred vibrations a second or more. Frequencies below this affect the integumental nerve endings and possibly the lateral line organs."

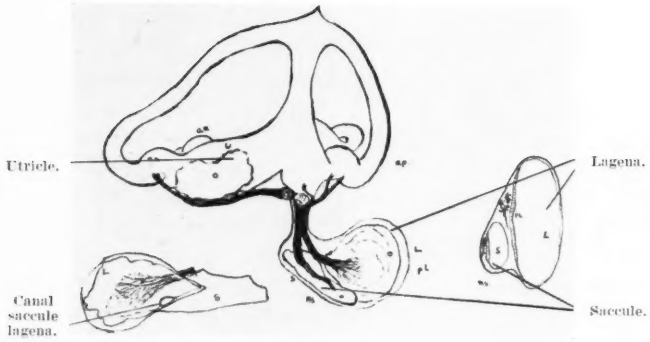

Fig. 4. Carp.

Large development of lagena in its relationship to the saccule. Canal of saccule lagena shown in left lower corner. Cross section of lagena showing relationship of lagena in right lower corner.

Fig. 5. Membranous saccule lagena of the lowest urodeles—Protein Anguinosa—Siren Lacertina. Relationship is the same as that in fishes.

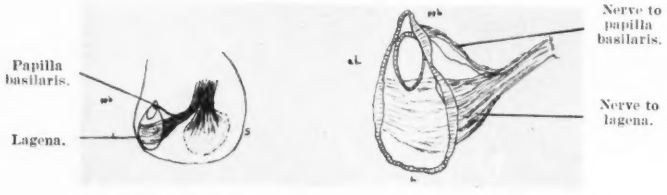

Fig. 6. Saccule and lagena of Siredon Mexicanus.

First appearance of the new neuro-epithelial sense organ in the region of the aperture of the lagena into the saccule.

Professor G. H. Parker also points out that in the hearing of fishes the saccula organ has nothing to do with equilibrium or muscle tonus, but is the chief organ of hearing, a function in which the sagitta (otolith) plays an essential part. (Structure and Functions of the Ear of the Squeteaque. 1908.)

Professor John Tait, in an article, "Is All Hearing Cochlear," states: "Notwithstanding these experimental complications, Mr. Ross obtained results which prove that both the lagena and the saccular macula of fishes are concerned with sound perception, though the precise partition of duty between the two receptors is still an unsolved question."

II. The amphibians show the first indication of a more specialized sense organ. In the lagena, near its opening into the succule, there appears a small sensory epithelial growth called the papilla basilaris. This papilla has its own nerve supply, and in the lower urodele is very small. In the highest of the amphibians, frogs and toads, there is a beginning encroachment of the lagena by the papilla basilaris with its surrounding structure, the basilar membrane. Fig. 5—Drawings of the lowest urodele family (Protein angunosa, Siren lacertina). Here, there is no evidence of the pars basilaris as these salamander-like animals do not leave the water during their existence. Fig. 6—Siredon mexicanus, a higher urodele with the beginning of the pars basilaris and papilla basilaris in the lagena. Fig. 7—Menopoma Alleghaniense (salamander-like) show a similar relationship. Fig. 8—Bratrachia, toads and frogs show a definite papilla basilaris, pars basilaris and its relationship to the lagena.

With the vertebrate beginning to leave its aquatic surroundings for an air environment, there is need for a new or more specialized sense organ for the perception of not only low frequencies but also high frequencies. The response of the air or partial air-living vertebrate to sound is not necessarily an interpretation or analysis of the sound but may be a conditioning reflex.

Thus in the amphibians there is direct evidence of a new neuroepithelial receptor developed for hearing in air. There is also a beginning perilymphatic system and middle ear development.

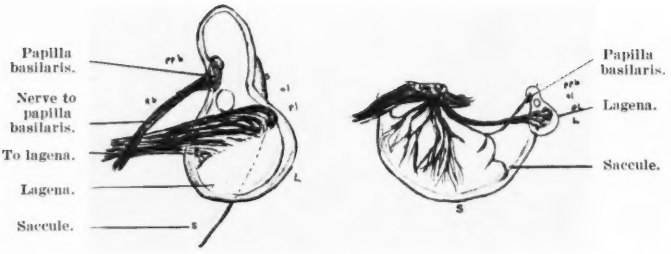

Fig. 7. Menopoma Alleghanniense.

Showing relationship of papilla basilaris to the lagena and to the saccule.

Fig. 8. Highest of Amphibians—Toad—Frog.

Showing tendency for the papilla basilaris to form a separate organ in relationship to the lagena.

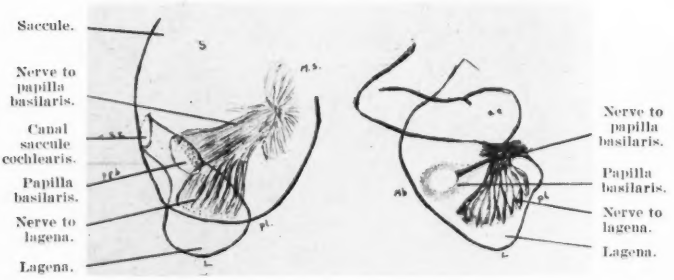

Fig. 9. Reptiles—Python—Snake—Chameleo.

In the chameleo the papilla basilaris and lagena are similar to that of the frog and toad. Python shows an enlarged papilla basilaris and its relationship to the lagena.

The reptilian group of vertebrates shows a gradual development to a higher form of cochlea. This is at the expense of the lagena, which is gradually pushed further away from the saccule toward the apex by the pars basilaris. There is now a definite cochlea with its basilar membrane, a perilymphatic system, a bony capsule and a middle ear development with its ossicle, the stapes (columella).

In the chamelo (Chamaeleon vulgaris) there is little difference from that of the frogs and toads (bratrachia)—Fig. 9. The python sebae (snake) shows a much higher development of the pars basilaris and the nerve of the cochlea is of much larger size.

In the lizard, the cochlea begins to assume the appearance of the mammalian cochlea with the lagena still occupying about half of the cavity. The green lizard has a splitting of the pars basilaris in two, and there is still more constriction and separation of the cochlea from the saccule—Fig. 10.

In the alligator the cochlea reaches its highest development for the reptile group. There is a definite cochlea now running the whole length of the bony capsule except at the apex, which still has the lagena. The cochlear nerve is large and the nerve to the lagena has become small, a reverse of the condition in the lower reptiles and amphibians—Fig. 11. Fig. 12 shows cross section of the lagena and cochlea in the alligator. The resemblance to the mammalian cochlea is great. The organ of Corti does not have a tunnel. There is a tectoral membrane, a scala tympania, scala media and scala vestibula. The lagena still has an otolithic mass in it and is in contact with the surrounding bony capsule.

Birds show an arrangement similar to that of the alligator. Fig. 13—shows relation of cochlea and lagena; Fig. 14, cross section of the cochlea and lagena.

III. The lowest of mammals, the monotremata, have a cochlea and lagena, the relationship being similar to that of the birds and higher reptiles, as the alligator. The organ of Corti now has a definite tunnel and a division of the hair cells into outer and inner groups. The lagena is similar to that of the bird—Figs. 15, 16, 17. Duckbill platypus (ornithorhynchis paradoxus). All other mammals of higher order have a cochlea similar to that of man. The lagena has disappeared, the cochlea having taken it

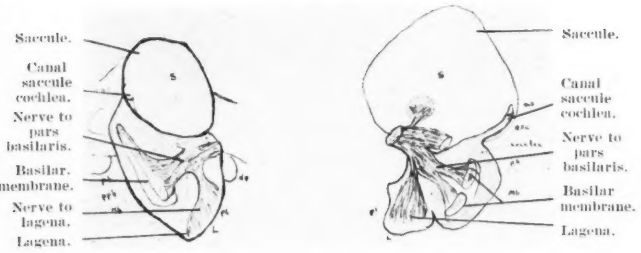

Fig. 10. Reptiles—Lizard—Green Lizard.
Showing elongation of pars basilaris and a beginning approachment to that of the mammalian cochlea.
Note: That in the green lizard the pars basilaris is split in two.

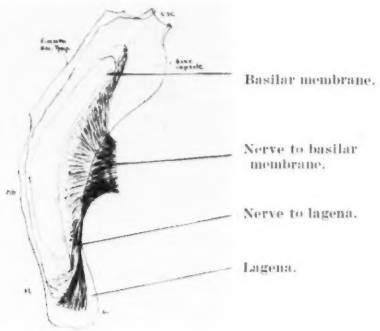

Fig. 11. Alligator.
Representing highest type of cochlea in the reptile group. Basilar membrane runs practically the whole length of the cochlea, and the lagena is now at the end of the cochlea furthest away from the saccule. Nerve to lagena has diminished greatly in size while that to the basilar membrane has increased.

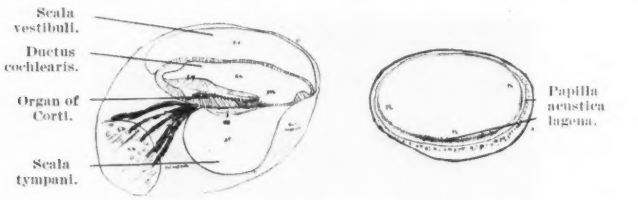

Fig. 12. Cross section of the alligator's cochlea showing the organ of Corti, basilar membrane, tectorial membrane, and relationship of the scala vestibuli and tympani to the ductus cochlearis.
Note: Cross section of lagena with the neuro-epithelium is in contact with the bony capsule.

Fig. 13. Bird.
Note: Marked similarity between the cochlea of the bird and that of the alligator.

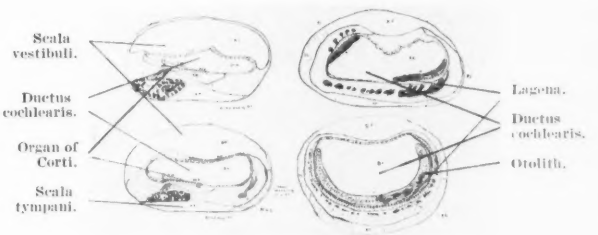

Fig. 14. Bird.
Cross section of cochlea and lagena.
Note: Structures are similar to that of the alligator. Lagena shows a large otolithic mass still present in its lumen.

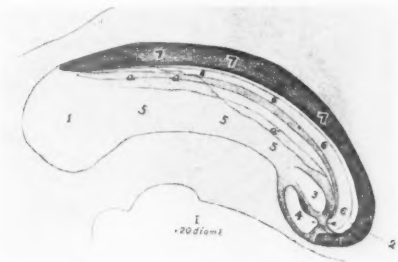

Fig. 15. Ornithorhynchis.
Drawings after those of U. Pritchard.
Longitudinal section of the duckbill's cochlea. 1. Opening into vestibule. 2. Apex of cochlea. 3. Helicotrema. 4. Cavity of lagena with arrow showing its connection with ductus cochlearis.

over completely. There is no longer an otolithic mass at the apex. The cochlea has become twisted and may be from a half a turn to that of five turns. Fig. 18—Cochlea of mammal rabbit.

W. Lorento de No, in an article, "Anatomy of the Eighth Nerve," calls attention to the following facts: That the nerve endings to the inner hair cells of the organ of Corti are specific—that is, the nerve endings are to one or two inner hair cells at the most. Of the outer hair cells in the organ of Corti he points out that the nerve endings from a ganglion cell are in connection with many hair cells and may run as far as a third of a turn or more in the cochlea. He then shows that the nerve supply to the basilar membrane is of a double manner and probably has double function; one for specific response, the other possibly for response to sound such as would occur in low frequencies.

R. L Wegel, "Excitation of the Auditory Nerve," states: "The 'low frequency range,' as here understood, is that bounded on its lower side by very slow fluctuations, 'zero' frequency, and at its upper side by that frequency below which the basilar membrane moves substantially as a whole—that is, all parts 'sensibly' in synchronism. It will be obvious that if a low enough frequency be chosen, say for safety, one vibration in ten seconds, .10 cycles, the basilar membrane must move as a whole. The same will be necessarily true for one vibration in 100 seconds, or .01 cycles per second. It will also be obvious that as the frequency is gradually increased from such a low value the shape of vibration of the basilar membrane, rigorously considered, undergoes gradual change. The question as to designation of the frequency at which this change in form of vibration becomes 'sensible' or perceptible has not been answered. It is provisionally identified here with the place, namely, between 100 and 500 cycles. No sufficient proof of this is offered, and it is subject to revision, probably downward, when further pertinent information is forthcoming." He later places it as low as 35 vibrations per second.

The conclusion of R. L. Wegel from a physicist's point of view is that the cochlea may respond as a whole to sounds of low frequencies.

Thus, between the primitive lagena and the complicated mammalian cochlea there is a gradual development, which can be

Fig. 16. Cross section of Organ of Corti of Duckbill's Cochlea. This is also after U. Pritchard.
Note: Tunnel of Corti which is not present in Reptile group. Division of hair cells into outer and inner hair cells.

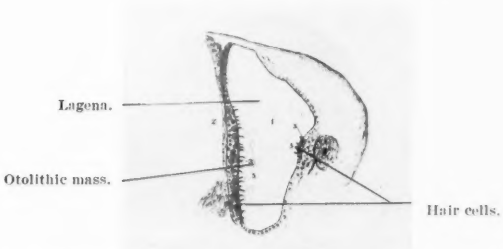

Fig. 17. Cross section of the lagena showing hair cells in the lagena and presence of otolithic mass. This is after U. Pritchard.

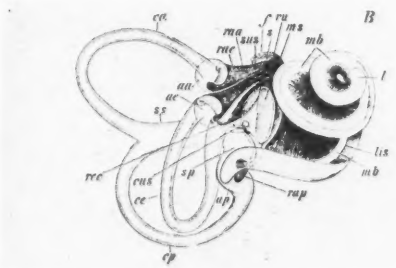

Fig. 18. Labyrinth of Rabbit. After Retzius.
There is no lagena present. Organ of Corti and membranous cochlea go to the tip of the cochlea. Extreme end of the cochlea is still called the lagena by Retzius. No longer an otolithic mass present in cochlea.

traced upward with scarcely a break. The lagena from its structure and its otolithic mass can only respond to sound as a whole. The mammalian cochlea, from anatomic, experimental and pathologic evidence, is capable of responding to sound from specific areas in the cochlea. From its phylogenic development the hypothesis can also be held that the cochlea can respond as a whole to sounds. The range of sounds to which the cochlea responds as a whole must be those that vibrate or move the cochlea as a whole. This must of necessity limit this type of response to the low frequencies or noises of low frequencies.

V. Conclusion.—From the phylogenic development of the cochlea one may conclude that the mammalian cochlea—

1. Is a development from two organs for hearing of which the lagena is the more primitive.

2. That with the cochlea taking over the function of the lagena it should respond in a similar manner as the lagena does.

3. The mammalian cochlea may therefore be capable of responding to sound in two ways:

(a) Stimulated as a whole in noise or low frequency sounds.

(b) Stimulated in specific regions for higher frequencies and musical tones.

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LXXXIV.

FIBRO-EPITHELIAL TUMORS OF THE NOSE (PAPILLOMATA) AND THEIR RELATIONSHIP TO CARCINOMA.*

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WICHITA.

There is so much confusion in reference to the classification of papillomas that when this term is used in the literature it is extremely difficult to indicate to just what pathologic picture one is referring.

The term papilloma has been applied rather generally to benign fibro-epithelial tumors of the epidermis and of the mucous membrane of the nose, bladder and other organs. Due to the aforementioned confusion of terms and because of the clinical and prognostic relationship of the fibro-epithelial tumors of the nose, in this paper the terms papilloma and fibro-epithelial tumor will be used synonymously.

It has been stated that papillomas of the nasal cavities and paranasal sinuses occur so rarely that each case should be reported, in hope that from these reports the accumulated data will help to solve some of the perplexing phases of this problem.

FREQUENCY AND LOCALIZATION.

Of 180,000 patients examined in Kofler's[1] surgical clinic, only three had papilloma of the nose. Moritz Schmidt[2] found 1,500 nasal tumors in 33,600 patients; of these growths, six were papillomas. In 1929, Eckbert Mobius[3] made a survey of the literature and found that sixty cases of papilloma had been reported. The majority of these tumors were the wartlike papillomas of the vestibule and the truly neoplastic papillomas of the deeper portions of the nose.

The scarcity of case reports is, at least in part, due to the fact that papillomas are frequently mistaken for polypi or other be-

*Presented before the American Laryngological Association, June, 1933.

nign growths, or, because of their malignant clinical manifestations, for carcinoma.

Fibro-epithelial growths arise as either solitary or multiple tumors, varying in size, form and consistency. They occur in greatest frequency in the vestibule of the nose, especially on the anterior portion of the septum. Here they spring from the transitional epithelium which lines this portion of the nose. Out of the thirty-two cases of papilloma of the nose which Hasslauer[4] found in the literature, twenty arose from the anterior portion of the septum. Papillomas also may arise in the posterior or superior portions of the nasal cavities, especially from the middle turbinal and ethmoid regions. They occur very rarely as primary growths in the paranasal sinuses. The following case reports are found in the literature: Herxheimer,[5] Holmgren,[6] Saxen,[7] Neumayer[8] and Ewing.[9] (Seydell, case No. 5.)

Papillomas are found more frequently in the male, usually between the fourth and fifth decade, but may occur at any age.

CLASSIFICATION.

In all these growths we are dealing with structures composed of both epithelium and vascular connective tissue, the supporting tissue being sharply demarcated from the epithelium covering it. It is therefore consistent to name them fibro-epithelial tumors. It will be noted that the quantitative relation between the epithelium and connective tissue varies considerably. In these fibro-epithelial (papillomatous) growths there remains in the tumor a definite type of structure, similar to the formation found in normal skin—i. e., a connective tissue stroma containing vessels and glands covered with epithelium.

There is no unanimity of opinion in reference either to the clinical or histologic character of the papillomas. Some authors classify them with the malignant tumors, naming them papillary carcinoma, adenoma malignum and adenocarcinoma; others hold that they are benign and name them adenoma, fibroma, fibro-epithelioma papillare and papilloma durum.

This diversity of opinion is due largely to the fact that the infrequency of these growths has made it impossible to make a comprehensive study of their histopathologic characteristics. In

some instances fibro-epithelial tumors of a papillary type lose some of their benign characteristics. In these cases it is extremely difficult to determine by a histologic examination whether the growth is benign or malignant. This fact will be referred to again later in the paper.

Papillary growths were formerly classified, according to the type of their epithelial covering, into hard and soft papillomas, the hard type having a surface membrane composed of stratified squamous epithelium, the soft type being covered with columnar epithelium.

It has been shown that this classification is not consistent with the facts, in as much as the squamous cell papillomas are frequently soft in consistency while those covered with columnar epithelium may be hard. The degree of firmness of the tumor depends upon the degree of development of the connective tissue and not upon the character of its covering membrane. It has been shown further that in some papillomas ciliated columnar epithelium may be found in immediate contact with stratified squamous.

ETIOLOGY.

In view of the fact that most modern investigators consider nasal papillomas as being on the border between benign and malignant growths, the etiology of these tumors will be discussed, including some of the causative factors that are common to both papilloma and carcinoma. The results of some recent investigations on the etiology of carcinoma will also be included.

It has been rather generally accepted that the essence of tumor formation and that of malignancy is within the tumor cell. The four theories most frequently accepted are:

1. Many neoplasms originate from misplaced embryonal cell rests—i. e., teratoma, neuroblastoma of the retina and sympathetic system, some of the angioma, etc.

2. There are undoubtedly many acquired tumors which have no relationship to embryonal cell rests. It has long been recognized that many new growths arise under the action of irritative factors. Usually this irritative factor is a prolonged one and may be of a mechanical or a chemical nature. Malignancies also

develop on the basis of chronic infectious processes and in old scars.

3. "It has been realized that the irritation theory of malignancy is inadequate. Many neoplasms arise where no history or evidence of trauma can be determined. In others the etiology is known, as in roentgen ray burns, and in these the removal of the causative factor does not stop the tumor formation. It has also been shown that a more or less prolonged period of time must elapse between the primary irritation and the formation of the new growth, which in turn signifies the presence of a continuous irritative process." (Fischer Wassels.)[10]

This investigator holds that the regeneration theory explains the development of these types of neoplasms.

The difference between the irritation and regeneration theories lies in the fact that neoplastic cells originate from normal cells through pathologic regeneration and that there is no direct connection between the external trauma and the formation of the tumor.

Further, Wassels believes that there must be two factors operating in conjunction to produce a malignant growth—i. e., the regeneration process and a constitutional factor. The nature of this constitutional factor is unknown. Also, it seems quite conceivable that this constitutional factor is essential in the development of those neoplasms which originate from misplaced embryonal cell rests. Although this theory has not been generally accepted by pathologists, many notable investigators support it in a general way and it seems to be gaining steadily in adherents.

ANIMAL EXPERIMENTS.

Fischer Wassels cites the following animal experiments to support his contentions. He experimented with mice by painting their skin surfaces with tar. In these animals no skin lesions were produced; but when a small area of skin that had not been previously treated with tar was burned, papilloma and squamous cell carcinoma developed. He also found that when a saturated solution of scarlet red was inoculated into the ears of rabbits, atypical epidermoid growths were produced resembling a precancerous lesion but never becoming malignant unless, for a con-

siderable period of time, the animal received small doses of arsenic. It was found necessary to continue the arsenic for from four to seven months before papillomas developed and from seven to eleven months before these tumors became malignant. (See Fig. 7.)

Clara Lynch[11] found that mice were very susceptible to spontaneous malignant tumors of the lung, varying in frequency with the strain of mice. When these mice were treated with tar, the malignancies increased from 7 per cent to 22 per cent in one strain and from 37 per cent to 85 per cent in another. Scabad[12] had similar results, and, in addition, found that in the offspring of mice whose ancestors had been treated with tar, the percentage of spontaneous carcinomas increased to a maximum of 74.3 per cent.

The presence of this so-called constitutional factor may explain why so many patients have embryonal rests, regenerative processes, papilloma and other benign growths, burns and scars, yet relatively few have cancers. It may also be the determining factor in those cases of papilloma which remain quiescent for years and then suddenly burst into malignancy.

A number of authors have investigated the possibility that some endogenous poison might be this so-called constitutional factor. Wassels administered indol to a group of animals and carefully observed their reactions. This experiment was carried on for a period of three years. The blood building organs of these animals became involved and a severe anemia with atrophy of the bone marrow resulted. Most of the animals died of an acute anemia. In those that lived, a reduction of the dosage produced regenerative processes followed by a late anemia. The survivors were now given still smaller doses of indol and developed aleukemic leukemia, lymphadenoma and myeloma; and some, metastasizing lymphosarcoma. Finally, those which did not succumb to these diseases developed a myelogenous leukemia.

4. Deelman, Lubarsch and others advance another etiologic theory which must be considered. They believe that tumors, especially the papillomas, which present for an indefinite period of time benign histologic characteristics and only later manifest malignant changes, do not undergo malignant degeneration or

pathologic regeneration, but actually possess, from the beginning, latent malignant properties.

In reference to the specific etiology of intranasal growths, we find in the literature infections and irritations listed as causative factors. It seems probable that they are only indirectly the cause of these tumors since many of the cases reported in the literature have not given a history of a discharge from the nose.

Many of the neoplasms spring from the deeper portions of the nose and from the nasal sinuses, a fact which in turn would seem to exclude direct irritation as a causative factor. To explain the cause of neoplastic growths, it would seem that here, as elsewhere in the body, one must choose between many theories and a few facts.

PATHOLOGY.

1. Macroscopic Pathology.—The fibro-epithelial growths of the nose may resemble ordinary polypi with the difference that the former are of a deeper red color and have a firmer consistency.

In other cases the growth appears as a series of deep folds in the mucous membrane (see case No. 5). Still others, and these have been termed the true papillomas or neoplastic growths, have a cauliflower appearance. These in turn may be divided into two classes—those arising in the deeper portions of the nose or nasal sinuses and those found in the region of the vestibule. The latter are much firmer and have a pale appearance resembling closely the ordinary wart.

There is still a difference of opinion as to whether in these tumors the primary hyperplasia begins in the epithelium or in the connective tissue stroma. It seems reasonable to suppose, as stated by Borst,[13] that in those tumors in which connective tissue predominates and there is a probability that they are of inflammatory origin, the primary development takes place in the connective tissue. On the other hand, where the epithelium in the tumor predominates, the primary hyperplasia more than likely occurred in the epithelium.

2. Microscopic Pathology.—Under low power magnification the non-neoplastic fibro-epithelial tumors present a papillary appearance in which the papillæ vary both in height and breadth.

The epithelium covering the connective tissue stroma may be composed of stratified squamous or columnar cells. In some tumors both types of cells may be found in the same section. Finally, the epithelium may be of the undifferentiated or atypical type. The epithelium is sharply demarcated from the connective tissue stroma and at no place should it penetrate the basement membrane. A varying amount of round cell infiltration is usually present. In these tumors the stroma exceeds the epithelial covering.

The cauliflower type presents slender stalks or budlike projections of vascular connective tissue stroma, varying in size and shape. The epithelium covering these projections is disproportionally massive, and may even reach 1 cm. in thickness. Here again we may find every variety of epithelial cells. "A few true papillomas have been reported whose covering epithelial cells consisted of a single layer of ciliated columnar cells." (Schmidtmann.)[14] These fingerlike projections extend deeply into the connective tissue stroma, but are sharply demarcated from it by a basement membrane. In some sections we may find that there has been a hyperplasia of the basal layer of cells which penetrates into the stroma, probably by pushing downward the basement membrane. Under low power one will find cross sections of these strands of cells as well as of every type of oblique section, depending upon the way in which the tissue has been cut.

Since many nasal papillomas are covered with squamous epithelium, it is natural to inquire how this epithelium originates in these locations. Its presence in the region of the vestibule has been explained by the theory that in areas where mucous membrane and skin come in contact, a transitional epithelium occurs in which the cells are not completely differentiated. In this area the cells therefore may develop into either the squamous or the cylindrical type. Where squamous epithelium is found in the deeper portions of the nose or nasal sinuses, we must look for some other explanation.

Herxheimer[5] has shown that many papillomas which were formerly thought to be of the stratified squamous type are in reality clothed with columnar cells which, in his opinion, have undergone changes in shape due to pressure caused by the rapid proliferation

of the epithelium. In these growths the microscope reveals that the upper layer of the epithelium is composed of closely packed cells resembling stratified squamous epithelium. The deeper layers contain cells which appear quite flat. They can, however, be differentiated from squamous cells by special staining methods. In addition, careful examination usually shows that in some of the valleys between the papilla normal areas of ciliated columnar epithelium can be found.

Some authors believe that squamous epithelium when found in the deeper portions of the nose is the result of embryonic cell rests. The presence of both ciliated columnar and stratified squamous epithelium in close apposition in the same section would make this theory unsatisfactory. This leaves one other explanation—i. e., metaplasia.

H. Renys,[15] who has made a thorough study of this question, thinks that pressure is not the causative factor in changing cylindrical to squamous epithelium, but strongly favors the metaplastic theory.

It long has been recognized that epithelium may undergo metaplastic change and it is more or less accepted that squamous cell carcinomas of the nose, bladder, bronchi and other organs develop on the basis of metaplastic epithelium. Listed among the causes of metaplasia are irritative factors, mechanical and chemical irritations, and chronic inflammatory processes.

Walback and Howe[16] found that metaplastic changes in the epithelium occurred when white rats and guinea pigs were fed a diet lacking in vitamin A. These changes were not confined to the eye where avitaminosis produces xerophthalmia but appeared in various organs. In the respiratory tract metaplastic changes occurred in the nose, nasal sinuses, trachea and bronchi. Similar changes were found in infants afflicted with avitaminosis.

CLINICAL SYMPTOMS.

Fibro-epithelial growths rarely produce severe symptoms. Nasal breathing gradually may become impaired, though years may elapse before complete nasal obstruction takes place. Secondary sinus infections occur when considerable nasal obstruction is present. Nasal hemorrhages are infrequent. Broadening of the

root of the nose, due to distention or destruction of the nasal bones, has been reported in a number of cases. (See case No. 1.)

DIAGNOSIS.

A diagnosis of nasal papillomas cannot be made on the basis of their macroscopic appearance. When destruction of tissue has taken place, papillomas easily may be confused with malignant neoplasms. Again they may resemble very closely the ordinary polypi.

Slow growth or absence of symptoms and of metastases point towards the benign character of the growth but cannot be relied upon as a diagnostic criterion. This is true especially since nasal carcinoma may remain quiescent for a long time without producing local or general symptoms, cachexia or metastases.

It is therefore essential to make a microscopic examination of a portion of the tumor. There should not be much difficulty in making a diagnosis by biopsy in typical fibro-epithelial tumors or if frank malignancy is present. Nevertheless, we must take into consideration the shortcomings of this method of diagnosis.

According to Reimann,[17] the microscopic diagnosis of malignancy does not seem to rest upon a broad scientific foundation in so much that pathologic interpretation of tissue is composed of 90 per cent of art and 10 per cent of science. Fischer Wassels[10] and others emphasize that empiricism plays the principal rôle in diagnosis by biopsy. Virchow's opinion that it is impossible to recognize a malignant tumor cell under the microscope still holds today. (Dietrich, Borst, Lubarsch, Sternberg, Maresch and Hertzler.)[17]

It is unfortunate that in regions of the body where skin and mucous membrane come in contact, tumors arise which may present an atypical epithelial arrangement. Under these circumstances, especially where it is possible only to remove a small portion of the growth, the histologic picture may be such that it is impossible to be certain whether the tumor is benign or malignant.

Another difficulty which arises to make the histologic examination uncertain is the possibility that a primary malignancy

may be associated with benign growths, or that a carcinoma may for a time present a papillomatous appearance.

The simultaneous presence of a papilloma and carcinoma may be accounted for in several ways. First, coincidence; second, both growths may have a common etiologic factor; third, a carcinoma may develop from a benign growth, as in the case of Bayer,[18] in which a polyp had undergone malignant regeneration at its distal end, the pedicle being free from malignant changes.

Renys reported a case in which a fibro-epithelial growth had been removed from the nose. The microscopic picture was definitely benign. (See Fig. 5.) Three months later there was a recurrence. When it was again sectioned, portions of it were found still benign while other parts were definitely malignant. (See Fig. 6.)

It is also true that no morphologic method has been devised whereby a conclusion may be reached as to whether a noncarcinomatous atypical cell proliferation will actually develop into a malignancy or will prove to be harmless. The deciding factor when such a lesion results in a malignant tumor may be an individual predisposition, the nature of which is utterly unknown.

Atypical papillary tumors are so numerous that the question of the advisability of the use of the terms precancerous or beginning cancer is rather important. It seems that the use of these terms as a histologic diagnosis is condemned by many of our best patholoists. "Ewing's writings indicate that he favors the theoretical conception of a precancerous stage of epithelial proliferation passing by insensible gradations from normal cells to fully developed cancer, but in practice he regards changes without definite signs of malignancy as benign and does not permit including 'precancerous lesions' in the statistics of cured cancer." (Helwig.)[17]

PROGNOSIS.

When not interfered with, fibro-epithelial tumors slowly increase in size. Some develop into large tumors, while others spread over a considerable portion of the nostril without forming a large tumor mass (author's case No. 3). Destruction of tissue and bone has been frequently observed, and in a number of cases

the papilloma has invaded one of the sinuses; thus the dura may be laid bare with meningitis resulting.

In a general way, the prognosis is more favorable if the tumor is solitary and if located in the anterior half of the nose. When the tumors are removed with a snare or punch, they recur in the majority of cases, frequently within a few months, the secondary growth often attaining a much larger size than the parent growth. These recurrences if removed will likewise return. Cases have been reported in the literature where recurrences have been removed over a period of twelve years without producing symptoms or becoming malignant.

It is unfortunate that we are not able always to be certain of the prognosis of papillomas. It is a well known fact that in papillomas of the bladder, which occur more frequently and which have been more thoroughly investigated than those which occur in the nose, malignancy frequently develops in histologically benign papillomas. This malignant change also occurs in papillomas of the nose.

"We are able to diagnose a fully developed carcinoma, but it is impossible to state that a benign appearing papilloma will not become malignant. The study of experimentally produced tar papillomas has shown that it is impossible to determine by morphologic methods which of a number of tar papillomas will later become malignant. In other words, a papilloma may be morphologically benign but biologically malignant." (Deelman.)[19]

Nasal papilloma which later have become malignant have been reported by Saxen,[7] Ewing,[9] Kesselbach-Hellman,[20] Pold,[21] Renys[15] and others.

TREATMENT.

In view of the fact that fibro-epithelial tumors of the nose are considered to be on the border between benign and malignant growths, it is essential that their treatment should not be too conservative.

When solitary tumors are found, removal of the growth with as much of its base as possible should be carried out. As an additional precaution against recurrences, the tissues from which the tumor sprang should be destroyed with a galvanocautery or diathermy.

Roentgen ray and radium have proved to be of great value in the treatment of all papillomas of the nose. Schiebe[22] effected a cure in a case of papilloma of the nose by using twelve exposures of X-ray of one-fourth erythemia dosage, in the course of eight months, supplemented with arsenic internally. With similar treatment Professor Brock[23] caused papillomatous masses in the nose to disappear. After fifteen years an examination showed that this patient had developed an atrophic rhinitis, but there were no recurrences.

A case reported by Renys[15] is very instructive. Histologically the tumor presented large branching processes covered with ciliated epithelium. On July 5, 1928, the tumor was removed and treated by X-ray radiation. In December, 1928, there was no recurrence of the growth. In April and again in June, 1929, small recurrences were removed. In these the epithelium had changed to a stratified nondifferentiated type without cilia and in addition the branching processes were much shorter. Towards the end of 1929 the epithelium had again returned to the original columnar ciliated type. In January, 1930, it was found that the tumor had invaded the frontal sinus. Here large branching processes with ciliated epithelium were found. A radical frontal sinus operation followed by X-ray therapy every month for three months effected a cure. There were no recurrences after a year.

In multiple tumors, all pathologic tissue should be removed to the bone or cartilage, followed by X-ray or medium therapy. If the sinuses are involved, external radical sinus surgery is indicated.

In the atypical or questionable cases, it is advisable to destroy the tumor by diathermy followed by X-ray or radium radiations.

Case No. 1.—Mr. O. J. G., aged 48 years. This patient came under the author's observation in July, 1926, on account of an external nasal enlargement associated with complete nasal obstruction. The enlargement had been noticed for about six years and was gradually increasing in size. Both nostrils had been completely obstructed for about four years. A nasal discharge had been present for sixteen years. Seven years prior to the author's examination some nasal growths had been removed on account of impaired breathing. There was no history of pain or severe

headaches. Laboratory tests, including a Wassermann reaction, were negative. A roentgen ray examination did not give the impression of tumor formation in the sinuses, although they were generally cloudy.

The examination revealed that the patient's nasal bones had been separated and pushed apart, the root of his nose being about one and one-half inches wide. The bone was completely absorbed in several places. The skin covering the nose was of a deep red color and had a pitted appearance. The facial appearance created was what the Germans term a *frosch gesicht* (frog face). Both nostrils were filled with firm reddish colored polypoid tumors. A large number of these were removed and the patient was instructed to return for observation but failed to do so. A letter of inquiry to his local physician in February, 1933, brought the following reply:

"Mr. G. is apparently well; his nose is still enlarged on both sides. I do not think it is more than one-third as large as it was, neither is it pitted and red as it was formerly." He further stated that the patient had had no trouble for about two years after the removal of the growths, but at the expiration of this time his nose gradually began to fill up again.

The report of the pathologist follows: "This section reveals marked proliferative changes in the epithelial lining. While in most areas the surface epithelium consists of four to six layers of high columnar cells with deeply stained slender nuclei, there is one area with deep infoldings of the epithelium, covering a formation of plump papillæ. These are lined with eight to twelve layers of plump cells with round or oval nuclei. The latter are lightly stained, vesicular, and have definite nucleoli. Only the basal layer consists of low columnar cells. These are well oriented and do not penetrate through the basement membrane. The majority of the cells in the papillary area are transitional. No prickle cells are found and no tendency to hornification is noticed. The stroma is a loose connective tissue with a few spindle and star shaped cells. There is marked edema and infiltration with eosinophiles, leucocytes, lymphocytes and plasma cells. The mucus glands are hyperplastic.

"Diagnosis: Adenofibroma edematodes (Ewing) with papillary proliferation of the transitional epithelium." Dr. Helwig.

Case No. 2.—Mr. O. C. E., aged 64 years. Was examined on March 31, 1924. His chief complaint was impaired hearing. In the examination of his nose a papillary tumor was found which sprang from the lower turbinate on the right side and extended across the floor of the nose to the septum. The growth was removed by snare and punch but no histologic examination was made. The patient did not come under our observation again until he was asked to return to the office for examination on October 10, 1932, when it was found that the growth had recurred. It presented a picture similar to that at the time of his operation in 1924. The papillary tumor was again removed and sections were made. The report on the histologic examination follows:

"The stroma of this section consists of connective tissue which varies in its consistency in different portions of the growth. In places it is loose and myxomatous in type; in other parts it is densely fibrous. It contains numerous well-formed blood vessels of various size. Embedded in the stroma are numerous glands which are typical of the mucous membrane of the nose. The papillary projections on its surface are broad and lined with stratified cylindrical epithelium of various thickness whose cilia have been lost in portions of the tumor. Here and there is found a proliferation of the deeper cell layers with consequent penetration into the stroma. In these places the epithelium loses its columnar shape and resembles the stratified squamous type.

"Diagnosis: Fibro-epithelial tumor." Dr. Jager.

Comment: The clinical term which has been applied to this tumor is papillary hypertrophy. The author believes insomuch as this tumor recurred after removal, it should be treated on the same basis as a true papilloma.

Case No. 3.—Mr. T., aged 72. This patient was brought to the office in June, 1931, by Doctor H. P. Knowles for a nasal examination. The patient complained of impaired nasal breathing and a sensation of pressure across the bridge of his nose.

In his left ethmoid region was found a cauliflower-like tumor mass which had destroyed most of his middle turbinate and ethmoid cells. The tumor was soft and bled freely when disturbed.

A clinical diagnosis of malignancy was made and a portion of the growth removed for biopsy. The histologic findings and diagnosis were as follows:

"The growth has an exquisitely papillar structure and presents budlike projections of vascular connective tissue stroma. The epithelium covering this growth is the stratified squamous type, the outer layer of which is more or less cornified. The stroma is more or less infiltrated with lymphocytes and leucocytes. The epithelium is everywhere sharply demarcated from the stroma.

"Diagnosis: Papilloma." Dr. Jager.

The patient refused to submit to radical surgery. He was asked to return for examination in October, 1932. At this time it was found that there was an extension of the growth in the ethmoid region and an involvement of the upper portion of the septum. His breathing was impaired but he complained of no other symptoms. There was no change in the histologic picture.

Comment: The author classifies this tumor as neoplastic papilloma. Its location prevents removal by radical surgical means, roentgen ray or radium ray treatment being the method of choice.

Case No. 4.—Mr. R. C., age 18. The patient complained of impaired breathing through his left nostril. Some growths had been removed from this nostril about one year ago.

Examination: Pus was found in both middle fossæ. A large polypoid growth protruded from beneath the left middle turbinate. Both antra and the left frontal sinus were flat on transillumination. This patient was kept under observation for a short period of time and was then advised to have a double radical antrum operation. This was carried out on March 29, 1921.

The tissue which was removed from the antra was not examined until March, 1933.

The pathologist's report follows: The tumor is a papillary growth. Slender stalks of loose connective tissue of considerable height are covered with several layers of columnar cells. The cilia of the uppermost cells are well preserved. The slender cells are lying in from two to five rows. They are of uniform size and staining reaction. No mitotic figures are found. The basement

membrane is well preserved and nowhere can there be noticed an invasion of epithelial elements through the basement membrane.

A few areas of the tumor have a smooth surface without definite papillæ, the epithelium being much lower in these areas. A group of acini with mucus filled cells are found in one area. The stroma is loose, edematous and large spindle and star shaped cells are scanty. There is diffuse infiltration with lymphocytes, plasma cells and a few eosinophiles.

Diagnosis: "Papillary fibro-epithelioma of the antra." Dr. Helwig.

Comment: This tumor is primary papilloma of the antrum.

Case No. 5.—Mr. C. B. R., age 31. On November 16, 1932, Mr. C. B. R., age 31, came in on account of a severe head cold. In his left nostril a growth was found which was about one-fourth of an inch in diameter and slightly more in length. Its surface was smooth and the tumor fluctuated. After the nasal infection had subsided, the growth was punctured and found to contain pus and granulation tissue. The latter was removed with a curette and sent to the pathologist, who reported as follows: "The section shows granulation tissue which is very necrotic; it stains poorly; there are no tubercles. By January 15, 1933, the growth had flattened to a papillar mass with a fistulous opening from which I could express a few drops of bloody, purulent fluid. So much of the tumor as was possible was removed and a section of the tissue was submitted to a number of pathologists of international reputation. One reported "the slide as a grade one adenocarcinoma of the mixed tumor type." Another stated that "the tumor stroma consisted of edematous connective tissue in which are found many large dilated blood sinuses. This tissue is covered by an irregular papillar growth of somewhat atypical nasal epithelium. This epithelial layer is very irregular and has some of the features of carcinoma, but I do not find any definite infiltration. It is possible that the whole process began as a localized cavernous angioma over which the epithelium has become disordered by infection. I am unwilling to make a diagnosis of carcinoma on this material. I think, if the bleeding area were cauterized or removed by diathermy, a cure would probably result,

but I would watch the patient carefully for possible carcinoma. There is a definite possibility that this is an early superficial carcinoma."

Comment: This growth is the type referred to in the paper as an atypical fibro-epithelial tumor. This patient has been under close observation since the removal of the growth. The wound has healed completely and there is no evidence of recurrence. The author has hopes that the tumor is benign but will keep the patient under close observation.

Case No. 6.—Mr. G. R. E., aged 41. Came to the office in February, 1932, on account of impaired hearing and tinnitus. On examining his nose, the author found a papillary growth on the anterior end of the left side of his septum.

This tumor was hard and wartlike in appearance, and while the patient was conscious of its presence he was not inconvenienced by it in any way.

The pathologist's report was as follows: "The tumor presented for histologic examination showed almost exclusively an epithelial structure of the squamous type, with atypical proliferation of the epithelium and marked cornification of the cells. It also contains numerous epithelial pearls. The stroma, which is very scant in this section, consists of fibrous connective tissue which is more or less degenerated. The diagnosis is epithelioma." Dr. Jager.

Comment: This growth is a neoplastic papilloma arising from the transitional epithelium of the vestibule. Although there is no evidence of malignancy from a clinical standpoint, the pathologist's diagnosis of epithelioma compels the author to regard the growth as belonging to those referred to previously as atypical and he has treated the case accordingly.

Case No. 7.—Mr. H. R., aged 54. This patient came to the author's office on December 17, 1932, complaining of severe pain over the left side of his head, frequent nasal hemorrhages, inability to breathe through his left nostril and a continual discharge from this side of his nose. The pain of which he complained had been gradually increasing over a period of eight months. The obstruction in his left nostril had been present for a much longer time. The patient was uncertain when it began.

The examination revealed a papillary growth which arose from the posterior one-third of his left lower turbinate and extended into and involved the upper portion of the nasopharynx. The histologic examination was as follows:

"The tumor shows definite papillary projections. The epithelium covering the papillæ is of the squamous type and presents marked atypical proliferation. The cells are hypochromatic and some are multinuclear.

"Diagnosis: Papillary carcinoma." Dr. Jager.

Comment: This case has been included to demonstrate a typical papillary carcinoma of the nose. As this patient had not been examined prior to the time the carcinoma was found, there is no evidence to show that the growth originated from a nasal papilloma.

CONCLUSIONS.

1. The infrequency of nasal papillomas is due in part to the lack of histologic examination of nasal tumor tissue.

2. Many investigators have associated ordinary polypi with the development of nasal malignancy. It seems probable that many of these cases have been papillomas which subsequently developed into malignant tumors.

3. It is not difficult to differentiate microscopically between typical polypi, papillomas and malignant tumors of the nose. At times the structure of these growths may gradually merge, making it impossible to diagnose by morphologic methods the true nature of the tumor.

4. Papillary fibro-epithelial tumors of the nose, especially the so-called neoplastic papilloma, should be considered on the border between benign and malignant growths. Clinically, therefore, they must be regarded with suspicion. Their treatment must not be too conservative.

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Fig. 1 (Case 1).—Showing external nasal enlargement.

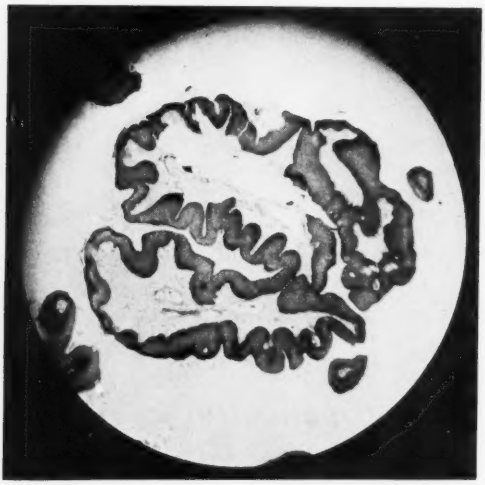

Fig. 2 (Case 3).—Neoplastic papilloma of the nose covered with stratified squamous epithelium.

Fig. 3 (Case 4.)—Neoplastic papilloma of the antrum covered with stratified columnar epithelium.

Fig. 4 (Case 5).—Papillary nasal tumor covered with atypical epithelium.

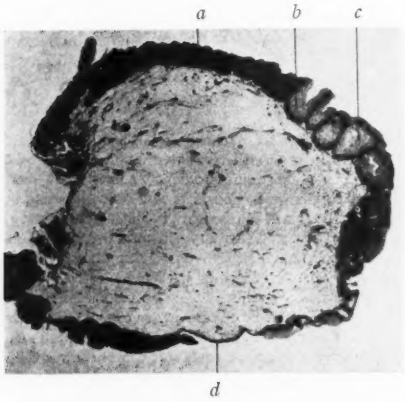

Fig. 5.—Benign fibro-epithelial tumor showing *(a)* stratified transitional epithelium, cilia being present in some areas; *(b)* stratified squamous epithelium; *(d)* stratified columnar ciliated epithelium. (Case of H. Reny's.)

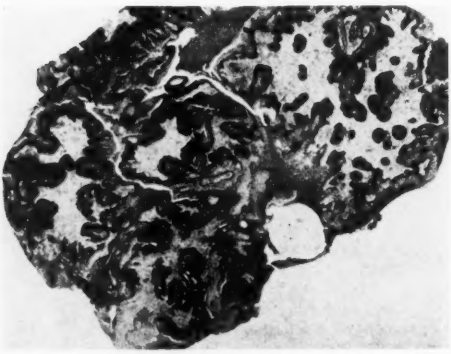

Fig. 6.—Both the papillary carcinoma and the benign fibro-epithelial tumor shown in Fig. 9 were removed during the same operation. (Case, H. Reny's.)

Fig. 7.—At the left is shown a typical skin papilloma which developed four weeks after the investigator had burned the skin of a mouse previously treated with tar for eleven months. On the right is an epithelioma in the same mouse. This developed four weeks after he burned the skin area, the mouse having been treated with tar for thirteen months. (Case, Fischer Wassel's.)

LXXXV.

PRIMARY CARCINOMA OF THE LUNG: BRONCHOSCOPIC OBSERVATIONS.*

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Most of the earlier writers considered carcinoma of the lung to be one of the rarest forms of pulmonary disease. At the present time it is generally admitted to be a fairly common condition. There has been much interest taken in this subject during the past few years and reported cases are increasing at an astounding rate. It is significant to note that in a twenty-four-year period, from 1852 to 1876, Reinhard[1] was able to find only five cases of primary carcinoma of the lung in 8,716 autopsies performed in Dresden.

At about the same time, from 1854 to 1885, Fuchs,[2] in Munich, could only find eight cases in 12,307 autopsies.

Even as late as 1912, when Adler[3] presented his excellent review of this subject, he was able to tabulate ninety cases of sarcoma and 374 cases of carcinoma of the lung.

When comparing these earlier reports with more recent statistics, it is evident that there is an actual increase in the occurrence of this disease.

Simpson[4] showed in his report, from the London Hospital, that there was an increase from 0.51 per cent in 1907 to 2.05 per cent in 1925, of tumors found at autopsy, and a comparative study showed a percentage increase of from 1 per cent in 1907 to 3.7 per cent in 1925, in primary carcinoma of the lung to all malignant growths observed.

A recent report[5] from the Pathological Institute of the German University of Prague is very interesting. From 1925 to 1931, 7,855 autopsies were performed, and in this group there were eighty bronchial and lung cancers. Comprising 1.02 per cent of

*Presented before the American Bronchoscopic Society, Washington, D. C., May 10, 1933.

all cases and 10.85 per cent of all carcinomas found at autopsy, this was a marked increase over the 1920 to 1924 group.

Seal[6] is of the opinion that primary bronchiogenic carcinoma comprises 5 per cent of all cancers and is almost one-third as common as gastric carcinoma.

While it is admitted that there is a real increase in this disease, many more cases have been found because we are becoming "cancer conscious," and they are now looked for more carefully.

We obtain more autopsies, the signs and symptoms are better understood and X-ray technic has improved. The most important of all has been the more general use of bronchoscopy in obtaining more information regarding the nature of this lesion. With a bronchoscope, early pathologic changes can be visualized in the bronchial mucosa, and if a suspected new growth is observed, a piece of tissue can be removed for microscopic study, a procedure which is the most valuable aid in making a correct diagnosis.

Jackson[7] stated: "Bronchoscopy is the only means by which an early diagnosis of endobronchial new growth can be made. By bronchoscopy the diagnosis can be made not only early, but with all the certainty of modern histologic methods."

Willy Meyer,[8] Lord,[9] Clerf and Funk[10] claim that between 85 and 90 per cent of primary carcinomas of the lung are bronchiogenic in origin and are usually found near the hilum of the lung, a location which makes them accessible for bronchoscopic study.

Vinson[11] has recently reported seventy-one cases of malignant disease of the lung observed at the Mayo Clinic, from May, 1925, to January, 1931. In all of these cases a diagnosis was made by removing a piece of tissue from the lumen of the bronchus and having it confirmed by microscopic study, a record to be proud of, and which demonstrates the accuracy and importance of bronchoscopy in recognizing this condition.

In a review of the literature on this subject, one is impressed by the great amount of attention which has been devoted to a study of clinical symptoms, signs, pathologic reports and X-ray observations.

All of which offer little definite help in making a positive diagnosis, whereas, bronchoscopy is the most important and most

accurate aid of all, but is given scant recognition unless the article is by a specialist in this field.

Therefore it is necessary for bronchoscopists to stimulate more general interest in this subject and to make the internist appreciate the value of this procedure in making a more correct and early diagnosis.

There has been considerable controversy over the relationship of pulmonary tuberculosis and lung cancer. Various authors estimate that the association occurs in from 5 to 20 per cent of all pulmonary malignancies. The process is not clearly understood. It has not been determined whether tuberculosis is activated by cancer or is inhibited in a carcinomatous condition.

Derischanoff,[12] at Roster, collected ninety cases of pulmonary cancer, of which 16.7 per cent showed a combination with active tuberculosis.

Recently an interesting case of this type came under my observation.

Case 1.—This patient, Mr. C., age 57, was sent to the Sanatorium with suspected tuberculosis. He had lost weight rapidly, had a chronic cough and brought up large amounts of bloody exudate. An X-ray plate revealed dense consolidation in the right lower lobe of the lung. No tuberculous cavities were made out and other parts of the lung appeared normal. Repeated examinations of the sputum did not show any tubercle bacilli. Later, X-rays and physical signs showed a rapid increase in the size of this growth and cancer was suspected.

A bronchoscopy was requested and examination revealed a large carcinomatous mass in the right bronchus. A microscopical section showed squamous cell carcinoma.

This patient finally died and an autopsy revealed not only extensive carcinomatous changes in the right bronchus and lower lung, but, to our surprise, numerous small tuberculous cavities were scattered through other parts of the lung which we had considered normal. This was a case in which cancer and tuberculosis were definitely associated.

Case 2.—Mr. F. F., age 47, gave no family history of tuberculosis, nor malignancy. He had lost thirty pounds in weight in a period of three months, during which time he had an intermittent cough. Physical signs were entirely negative and nothing definite was made out by X-ray examination. At the time of his death an autopsy showed carcinoma at the right hilus of his lung associated with an old healed tuberculosis.

Case 3.—Mr. J. K., age 52, was just the opposite because he had a definite family history of tuberculosis and he had physical signs of extensive involvement of the right middle and lower lobes. This was confirmed by X-ray plates. On bronchoscopy, a carcinomatous mass

could be made out, occluding the right bronchus and causing atelectasis of the lung. A biopsy showed the "oat" cell type of carcinoma.

Autopsy examination revealed considerable ulceration in the bronchus and bronchiectasis, with marked infiltration of the lung structure.

Most of the cases of carcinoma of the lung, run a rather rapid course, lasting from two to twenty-four months as a rule, so the following case is of unusual interest because a squamous cell carcinoma was discovered in this patient over seven years ago and he is still living.

Case 4.—Mr. K., age 45, was examined by Dr. Jackson about seven years ago at which time he was informed that he had a cancer of the left bronchus.

He consulted me last March, complaining of losing weight, coughing up great quantities of blood and purulent exudate. Bronchoscopic examination showed a fairly large tumor mass in the left lower bronchus. A section of the growth was taken and reported to be squamous cell carcinoma. This patient refused radium treatment, but with endothermy and X-ray, the growth was reduced in size and the hemorrhages controlled. This patient gained weight, much of his exudate subsided and he is still living and getting along fairly well.

The next case was one in which the course was very rapid and presented a number of interesting features.

Case 5.—Mr. R. T., age 41, came to me with a history of suddenly coughing up a large quantity of blood without other symptoms.

A laryngeal examination showed fresh blood on the posterior laryngeal wall. Tuberculosis was suspected and he was immediately referred to a specialist in this disease. No physical signs of tuberculosis were found; X-ray examinations were negative and no tubercle bacilli could be found in the sputum.

A bronchoscopy was performed but no new growth nor cause for the hemoptysis could be found.

He went along for about six weeks without any symptoms and had no further hemorrhages. He then came to my office complaining of epileptic attacks of short duration with loss of consciousness. Vision was found to be normal, eye grounds were normal and no changes in the visual fields could be made out.

During a period of ten days his symptoms became rapidly worse, his vision was reduced to 20/200 and visual fields became concentrically contracted to ten degrees and a diagnosis of brain tumor was made.

An operation revealed a small metastatic tumor in region of supramarginal and supra-angular gyri on the left side. Patient died after the operation and an autopsy revealed a small carcinoma in the left lower bronchus, which was the primary lesion.

Cases with metastatic involvement of the brain are comparatively rare, particularly with such a rapid course. Evidently the metastasis was carried directly through the blood stream.

This case is interesting because it demonstrates the importance of spontaneous hemoptysis in adults as a cardinal symptom of carcinoma of the lung in which tuberculosis can not be found.

Case 6.—Mr. L., age 54 years, was treated for several months with opiates to relieve an obstinate bronchial asthma. Considerable dyspnea was present most of the time. At no time during the early treatment was a new growth suspected in the trachea until patient changed physicians. Dr. Cullen at the State Sanatorium examined this patient and considered the possibility of a new growth.

A bronchoscopy was made and a rather large intratracheal new growth was discovered just above the bifurcation of the left bronchus. This growth was reduced by endothermy and the patient obtained instant relief. He went along for several months very comfortably but eventually had a recurrence with metastatic involvement of the mediastinum. This patient could have been saved much suffering if a new growth had been discovered earlier by bronchoscopy and proper treatment instituted.

Case 7.—Mr. B. G., age 77, was admitted to the Hospital complaining of loss of weight, constant cough and some difficulty in swallowing. Patient was given a barium meal for X-ray examination and the solution passed into the bronchi as shown in the lantern slide.

Bronchoscopic examination showed a fistula between the esophagus and the trachea just above the bifurcation, due to a carcinoma.

This case is shown especially because it shows how well the bronchi are outlined by the barium solution, and the fistula between the trachea and esophagus is uncommon.

Case 8.—This case is presented because it shows very clearly by lantern slides the affect of radon implants on a carcinoma of the lung.

The first slide shows the growth at the time of the first treatment, while the second one demonstrates the appearance of the lung two weeks later and it is very noticeable how much the growth has decreased in size. During this time the patient has gained twelve pounds in weight, hemorrhages have stopped and he feels very much better.

In reference to treatment, we have had no cases suitable for lobectomy or any operative procedure.

We have depended upon endothermy, radium and X-ray to alleviate the symptoms. These agencies are of value particularly in controlling hemorrhage and reducing the size of the growth.

The bronchiectasis can be helped by suction and local treatment, which tends to make the patient more comfortable.

SUMMARY.

1. Primary carcinoma of the lung has shown a definite increase in the past few years.

2. As 85 to 90 per cent of these cases are bronchiogenic in origin, the bronchoscopist has the best opportunity to make an early diagnosis and outline proper treatment for these cases.

3. Bronchoscopic examination, at which time a piece of the growth can be removed for histologic study, affords the most satisfactory method of making an exact diagnosis in these cases.

4. The importance of bronchoscopic examinations in obscure chest conditions should be given more attention.

5. Routine bronchoscopies in such cases at times reveal unsuspected and interesting lesions.

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LXXXVI.

OTOLARYNGOLOGIC CONDITIONS WRONGLY ATTRIBUTED TO AN ENLARGED THYMUS.

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Houston.

There has been an enormous amount written in regard to this subject, particularly the part played by the thymus in producing laryngeal symptoms. There is such a diversity of opinion that in reading on this condition one is left in a state of confusion. Too often cases with laryngeal symptoms are wrongly diagnosed as being due to an enlarged thymus and treated as such before a complete examination is made. Also in cases of sudden and unexpected death during or after anesthesia we are only too quick to conclude that it was due to an enlarged thymus or status lymphaticus without searching further for other causes.

The symptoms so frequently judged as being due to an enlarged thymus are: chronic stridor, hoarseness, attacks of choking, wheezing and occasionally cyanosis. It is my opinion that in the study of these cases not only should this consist of very careful general examination, including roentgenographic study of the chest and neck, but should also include a direct examination of the pharynx, larynx and trachea. There should never be any hesitancy on the part of the pediatrician in allowing this to be done, as it is a most simple procedure. Direct laryngoscopy is carried out without any preliminary preparation or anesthetic and can be completed in a very short time without any after effects on the patient.

In most of these cases the symptoms are present at birth or develop shortly afterward. There is usually noted an inspiratory stridor, although when marked there may be an expiratory grunt. Cyanosis is not frequently seen, but there is often some retraction of the sternum or costal interspaces on inspiration. These cases generally outgrow this condition with the development of the

larynx between six to eighteen months. It is uncommon to have a fatality and when it does occur it is usually brought on by some intercurrent infection.

New and Kennedy,[1] in describing chronic stridor in childhood, classified the causes into two main divisions: or those cases where the lesion is intrinsic or extrinsic. Of the intrinsic causes they believe the most common type are those cases where there is a congenital relaxation of the laryngeal structures. Other intrinsic causes frequently seen are those due to a bilateral abductor paralysis, tetany with resulting spasm, neoplasms, the most common of which is the papilloma and a subglottic diaphragm. In considéring the extrinsic causes, they describe mediastinal and cervical tumors which encroach on the larynx and trachea, lingual thyroid gland, unusually flaccid tongue and a retropharyngeal abscess, as seen in cold abscess of tuberculosis.

Hill[2] was one of the first to fully describe those cases where a congenital relaxation of the laryngeal structures are seen. He describes the most common picture as being that where the epiglottis is folded in more than normal and the aryepiglottic folds are thin, flaccid and in close approximation. There are many variations of these flaccid larynges, but it is generally conceded that in main there is simply an exaggeration of the normal infantile larynx. Another picture frequently seen in these types of cases is where the arytenoids are greatly elongated and unsupported and are easily drawn into the glottis on inspiration. The stridor being due to the vibrations of the folded epiglottis or arytenoids and the sucking in of the flabby, unsupported structures, I feel that it is in this type of larynx that the symptoms are most frequently attributed to an enlarged thymus.

Pancoast,[3] in a report on roentgenology of the thymus in infancy, states that no examination is complete without a careful pharyngeal and laryngeal examination. The study of the thymus must be made through observation of the upper respiratory passages rather than just a study of the gland itself. He feels that in the roentgenographic study of the thymus all patients should first be examined with the fluoroscope in both the sagittal and lateral directions. This study should include the neck and chest

and the effect of both phases of respiration noted. Roentgenographic exposures should be made in the erect posture, taking two pictures in each the sagittal and lateral positions: one, to be taken during full inspiration and one during full expiration. It has been my past experience that in a large percentage of the cases only one picture is taken, and that, with the patient lying down and this exposure made in the anteroposterior position. From this one picture a diagnosis of enlarged thymus is made and treatment instituted. It has been shown by Hasley and de Tomasi[4] the many sources of error that may arise in the ordinary roentgenographic study of the thymus. With their ciné-camera studies they were able to demonstrate how the thymic shadow changes during respiration and the effect of systole and diastole of the heart. Hasley states that the diastole, the systole, the gorgement at one time of the heart, the partial constriction at another time, the change in size of the pulmonary artery, aorta, inferior and superior vena cava, all exert different pressure changes on the thymus gland. This study would tend to prove the difficulty in making a diagnosis of an enlarged thymus from an X-ray exposure. This would further show that unless the films taken before and after roentgen therapy represent identically the same phase in the cardiac and respiratory cycle they have no significance whatsoever. This report puts some doubt on the accuracy of the many reports on reduction in size of the thymus following roentgen therapy.

An enlarged thymus is seen quite frequently in normal infants, and it is easy to understand why a diagnosis of enlarged thymus is so often made in infants with laryngeal symptoms. Liss[5] reports a large series of X-rays taken of normal infants shortly after birth, and in his series 42 per cent revealed a shadow suggesting enlargement of the thymus. None of these cases had any abnormal symptoms. On following these cases a retrogression of the thymus rapidly took place, the greatest change occurring in the first three months.

We frequently hear of the value of roentgen therapy in all preoperative cases where the thymus is enlarged. One of the most enthusiastic in this regard is Mosher.[6] In his clinic routine thymus pictures are taken on all preoperative cases and roentgen treat-

ment instituted in all cases where the thymus is found enlarged. He feels that this procedure has done away with the so-called thymic death in his clinic. However, Cooperstock[7] reports eight cases that had received X-ray treatment for thymic enlargement and subsequently had died during or after operation. Six of these cases came to autopsy, five of which showed evidence of status lymphaticus.

New and Kennedy[1] report that during a period of ten years no child was seen with symptoms which they felt were referable to hypertrophy of the thymus. In this same period of time no death occurred which was attributed to the thymus, either during or following anesthesia. In the Mayo clinic patients are not subjected to roentgenographic treatment of the thymus, preliminary to operation. However, a great deal of attention is paid to the preoperative care of the children showing evidence of any condition which might increase the risk of operation.

The selection of a competent anesthetist cannot be too strongly advised. Many anesthetic deaths which have been reported as being due to status lymphaticus have been caused by an overdose of the anesthetic used. Waldbott[9] recently reported several cases which had been termed thymic deaths, but in reality were anaphylactic reactions to the anesthetic. He feels that all allergic patients to be operated on should be tested beforehand for the anesthetic which is to be administered.

REPORT OF CASES.

Case 1.—B. B., age six weeks, was admitted to the St. Louis Children's Hospital, November 3, 1929. The chief complaints on admission were noisy respiration and attacks of cyanosis. Diagnosis of enlarged thymus had been made before admission and treated as such without relief of symptoms. This diagnosis, however, was not verified by the physicians in the children's hospital. There was a rather marked inspiratory stridor with some sternal retraction. Direct laryngoscopic examination revealed both arytenoids to be long and tapering and were drawn into the glottis on each inspiration. No treatment was instituted and the parents were advised that with the development of the larynx the symptoms would disappear. The patient remained under observation for five months and during this time the condition gradually improved and at the time of discharge the stridor had entirely disappeared.

Case 2.—C. R., age three months, was first seen November 18, 1931, with history of difficulty in breathing since birth. The mother also complained of the baby choking at each feeding time. There was a rather

marked inspiratory stridor which was greatly aggravated by crying or excitement. Roentgenographic study made shortly after birth revealed an enlargement of the thymic shadow. X-ray therapy was instituted and carried out without relief to symptoms. Direct laryngoscopic examination showed the epiglottis to be elongated and bent inward. Both aryepiglottic folds were enlarged and extremely flaccid. On each inspiration this flabby tissue was drawn into the glottis, causing partial obstruction. The pediatrician was advised to discontinue all X-ray therapy and told that with the development of this child's larynx he would be relieved of his symptoms. One year later when the baby was seen he was entirely relieved of his previous difficulty.

Case 3.—H. C., age twenty-three months, admitted to St. Louis Children's Hospital, August, 1930, with history of attacks of dyspnea and cyanosis since birth, which was progressively growing worse. General examination was negative. On direct laryngoscopy multiple papillomas were found arising from the larynx. These were removed by blunt biting forceps and had to be repeated on four different occasions. Following each removal the patient had complete relief from his symptoms and following the fourth operation the baby was still free of symptoms when seen five months later.

Case 4.—F. B. M., age eleven months, was first seen July, 1932, with history of difficulty in breathing since birth. Roentgenographic studies made shortly after birth revealed an enlarged thymus and X-ray therapy was instituted. The symptoms continued in spite of this treatment. With each fresh injection the symptoms became quite alarming and on several occasions tracheotomy was considered. On examination the child was found to have a marked inspiratory stridor with some retraction of the sternum. Direct laryngoscopy revealed the laryngeal structures to be relaxed with definite elongation of each arytenoid. On each inspiration these structures were all drawn into the glottis. This child was seen about two months later with an acute infection and was having considerable difficulty in breathing. Direct laryngoscopy this time showed this flabby tissue to be edematous and swollen, almost completely obstructing the glottis. The parents were told this child would outgrow this condition and were cautioned in regard to acute infections. At last report, or eight months later, the child was greatly improved.

Case 5.—J. A., age seven years, entered St. Louis Children's Hospital, October 8, 1930, with a history of difficult breathing since birth. His symptoms were always aggravated by exertion or excitement. One year previous to admission to hospital his breathing became so labored that tracheotomy was found necessary. When first seen the patient was still wearing the tracheotomy tube, on closing this he had considerable difficulty in breathing, having a marked inspiratory stridor. Laryngeal examination revealed a complete abductor paralysis of both cords. It is interesting to note that the mother of this boy also had a bilateral abductor paralysis and a sister, who was not seen, also had difficulty in breathing.

Case 6.—This case was previously reported. C. K., age eight weeks, was first seen November 24, 1929. The chief complaints were dyspnea, cyanosis and difficulty in nursing. Previous to admission to the hospital a diagnosis of enlarged thymus had been made. On general examination there was found a malnourished infant with an inspiratory stridor, receed-

ing type of chin, wide cleft in the hard palate and marked sternal retraction. Direct laryngoscopy was attempted, but due to the fact that the base of the tongue was greatly enlarged and in close proximity with the posterior pharyngeal wall it was impossible to see the larynx. It was then noted that on retracting the tongue the breathing became normal, but on releasing the tongue it would fall back, blocking the oropharynx, causing the stridor to reappear. There were two factors in this case causing symptoms: first, the macroglossia, and second, the receding type of mandible.

CONCLUSION.

Any of the cases reported could be and several had been diagnosed as being due to an enlarged thymus, whereas, the lesion was entirely confined to the larynx and pharynx. For this reason, on every infant presenting laryngeal symptoms a direct laryngoscopic examination should be made. It is my belief that in the majority of these cases the etiologic factor will be found in the pharynx and larynx rather than the thymus, as is so often reported. The thymus shadow is found enlarged in over 40 per cent of normal infants. The usual roentgenographic studies of the thymus are inaccurate and are apt to give false impressions. The size of the thymus shadow varies in the different phases of the cardiac and respiratory cycles. I do not feel that deaths which occur during or following anesthetics could be attributed to an enlarged thymus. Postponement of operation and careful preoperative care of all patients who show evidence of upper respiratory infection, renal or cardiac disease, fever, marked anemia, malnutrition or any other condition which might increase the operative risk. The selection of a competent anesthetist cannot be too strongly advised. All allergic patients to be operated on should be tested for the anesthetic to be administered.

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LXXXVII.

SOME CLINICAL OBSERVATIONS ON NASAL HEMORRHAGE.*

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Nasal hemorrhage is not a myth. In the active practice of the rhinologist it is one of the frequent and by no means trivial occurrences in his routine work. It seems absurd to present a paper before this society on such a trite subject, and yet to the older men in the practice of rhinology, the writer knows of no subject which has brought more disturbing memories than the many hours in their past clinical life which were spent in attempting to relieve a patient suffering from nasal hemorrhage. Like Banquo's ghost, a severe epistaxis frequently occurs during the dreamy hours of night, when man's slumber should not be disturbed by such a rude awakening as a call to stop a severe nasal hemorrhage. But all of us have had our troubles, and it is just the commonplace things in life which give us the most concern.

Several years ago, the writer reported a case of uncontrollable nasal hemorrhage, followed by death, as the result of an intranasal ethmoidectomy, where the many sleepless hours engendered through our attempts to relieve this condition brought more than one wrinkled furrow to a once placid countenance.

A complete clinical and bibliographic discussion of this subject is beyond the scope of my paper, but the writer wishes to call attention to a very satisfactory and simple procedure which he has used for years in the relief of the ordinary spontaneous nasal hemorrhages, and especially those arising from the anterior part of the nasal septum.

Traumatic nasal hemorrhages and such as follow intranasal operations are not, as a rule, difficult to manage, because we anticipate that such may occur, and we take precaution to tampon

*Presented before the Fifty-fifth Annual Congress of the American Laryngological Association, Washington, D. C., May 8-10, 1933.

the bleeding area. Even then, in a subject having a tendency to bleed, the oozing which sometimes follows is often quite difficult to control.

The other class of nasal hemorrhages which give us the most trouble are those which occur in elderly people where the presence of an arteriosclerosis is a contributing and exciting cause for this condition. How frequently one hears it said that a nasal hemorrhage in this class of people probably saved them from an intracranial hemorrhage, and for that reason it probably was a life saver for that individual. However, this is a small comfort to the rhinologist who is called in to stop the hemorrhage and whose ability to accomplish the necessary results is taxed to the utmost. Packing of the nasal cavities in these cases is a hazardous procedure. Hazardous, not in the actual packing, but the troublesome hemorrhage when the packing is removed. This occurs in spite of the packing being saturated with some oleaginous material. Hemorrhage in these cases arrested without any packing is a far more desirable procedure.

In the writer's experience, the majority of nasal hemorrhages in elderly people and those with arteriosclerosis arise from the neighborhood of the middle turbinate and the attic of the nose. Rest in bed, especially where hypertension is present, is absolutely necessary in these cases. Temporary adrenalin packs and a spray of the same solution, together with horse serum or even the intravenous injection of the patient's own blood, has, in the writer's experience, proven to be the best line of procedure. Such cases must necessarily be under the strict supervision of the internist.

At the International Congress of Otolaryngology, held in Copenhagen in 1929, a very practical article was presented by Dr. Aspisoff of Tiflis, in which he described his operation for the cure of nasal hemorrhage when the same occurred at the usual bleeding point on the anterior end of the nasal septum. His procedure was as follows:

After cocainizing and adrenalizing the nasal mucous membrane, an oval incision about 1 cm. from the bleeding point is made posterior and above, down to the cartilage, in order to cut

through the bleeding vessels. The bleeding surface is now covered with a sterile gauze tampon, saturated with peroxid of hydrogen, and allowed to remain for twenty-four hours. After healing, there remains a fine, hardly perceptible scar at the point of incision, while the blood vessels below gradually shrink and disappear. The bleeding disappears immediately and remains permanent.

Aspisoff has used this method for many years with unusually good results. The present writer has tried this method in a few cases, but cannot say that it has been universally successful. According to Aspisoff, two-thirds of the cases of nasal bleeding occurred from the anterior part of the nasal septum, ten per cent from the floor of the nose, and 15 per cent from the posterior part of the nasal cavities. I am entirely in accord with this writer when he says that the most difficult cases of epistaxis to manage are those occurring in elderly people with arteriosclerosis. Nasal packing in these cases is to be avoided, if possible, on account of recurrent bleeding when the packing is removed. Every abrasion will start a new point of bleeding. Long nasal packing is apt to produce infection which may extend to the sinuses or middle ear.

Any simple procedure is certainly to be welcomed, especially if it is efficacious.

A number of French and German rhinologists have reported good results from the use of electrocoagulation in all kinds of nasal hemorrhage. The present writer has also used this method. My objection to it is the fact that the cartilage is frequently injured and is sometimes followed by scabs and even perforation.

In 1902, Dr. Charles H. Cargyle, in conjunction with Johnson & Johnson, and with the aid, also, of Dr. Robert T. Morris of New York, and Dr. John B. Deaver of Philadelphia, succeeded in producing an animal membrane which has since been known as the Cargyle membrane.

The membrane finally selected was made from the peritoneum of the ox. This membrane is sterilized and placed in hermetically sealed envelopes. It was found that this membrane, placed in contact with the tissue surface, became adherent at once, and thus upon an abraded surface, it was not easily displaced, form-

ing a protective that would prevent adhesions and itself be absorbed in a varying course of time. Consequently, it has been used in intestinal operations, skin grafting, burns and to a considerable extent in brain surgery.

Six years ago the writer started the use of this membrane in those cases of denuded membrane with scabs so often seen on the anterior portion of the septum, and from which the large majority of nasal hemorrhages occur.

The method of using this membrane in such cases is as follows:

After controlling the bleeding by tampons of adrenalin or peroxid of hydrogen, the point or points of bleeding are touched with a 4 per cent solution of nitrate of silver. Over this is spread a layer of Cargyle animal membrane, just large enough to cover this area. The membrane will adhere perfectly to the septum surface, and if not, flexible collodion can be used. The patient is told not to blow the nose for twenty-four hours. At the end of this time, if the membrane is loose or has disappeared, another layer is put in the same position. This has to be repeated perhaps several times.

In addition to the use of this membrane in those cases of hemorrhage from the septum where there is no perforation, the writer has found its use most valuable in those cases which are sometimes seen accompanied by large perforation of the septum accompanied with considerable scabbing and bleeding from the exposed edges. We mean, of course, in those cases where the perforations are large and where it is almost impossible to rectify the same by surgery. It is here that the membrane can cover the edges and obtain comfort for the patient where all other methods have failed. The same procedure is adopted as one uses it in the milder forms of bleeding, thus preventing a further disintegration of the cartilaginous septum. Just what is the biochemical action of the Cargyle membrane in these cases is difficult to say, but certain it is that it aids very materially in the regeneration of the matrix cells both in the mucous membrane and the cartilage itself.

In all my cases there has been no return of the bleeding, and this has extended over a period of several years.

In other portions of the nasal cavity, the same procedure can be used with the exception that it is more difficult to place this

animal membrane over surfaces which are not quite so accessible. However, tampons can be made of the same membrane and used just as readily as any other material. In those cases of hemorrhage due to arteriosclerosis these membranes are far superior to other packings in that one need never be concerned about their removal. Hurd of New York is a strong advocate of placing strips of fat pork in the nasal cavities for arresting any intractable hemorrhage in this region. However, in a few cases of mine where it was used the results were not as satisfactory as desired.

In hemorrhage from the posterior portion of the nasal cavity, where it is impossible to see the bleeding points, we must resort to the postnasal tampon to obtain immediate results. In addition, however, the animal membrane placed as a tampon more anteriorly is a most excellent adjuvant.

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LXXXVIII.

A STUDY OF CHRONIC UNEXPLAINED COUGH WITH THE AID OF THE BRONCHOSCOPE.*

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BOSTON.

When chronic cough, with or without sputum, is not readily explainable by some such condition as tuberculosis, pulmonary abscess, foreign body, bronchiectasis, malignant disease, or heart disease, it is usually designated as chronic bronchitis. The purpose of this investigation was to study such cases with the aid of the bronchoscope to determine the presence or absence of abnormal changes in the bronchi. We also wished to observe the symptoms of such patients after aspiration of the abnormal secretion and instillation of lipiodol into the bronchi.

METHOD OF STUDY.

Twenty-five cases with chronic recurrent unexplained cough were studied for two years in the Medical and Otolaryngologic Clinics of the Boston Dispensary. In twenty cases there was productive cough, in two unproductive cough, and in three productive cough associated with bronchial asthma.

A history, complete general examination, urinalysis, blood study and sputum examination for tubercle bacilli were provided in each case. The nose, throat and accessory sinuses were examined. At a subsequent date the patient presented himself in a fasting condition for bronchoscopy. The pyriform sinuses were anesthetized with 10 per cent cocain solution, and a few drops of the same solution were introduced into the larynx with a laryngeal syringe.

The patient was then placed in a sitting position, and the bronchoscope was introduced. The bronchial tree was carefully

*From the Otolaryngological and Medical Clinics, and Department of Clinical Research, of The Boston Dispensary. This research was aided in part by grants from Tufts College Medical School.

inspected and any visible secretion was aspirated. Ten cubic centimeters of lipiodol were then introduced into each main bronchus, followed by a roentgenogram of the thorax. After a short rest, seldom exceeding an hour, the patient was sent home, to return at fortnightly intervals for observation.

Cultures of the aspirated secretion did not show growth in the first few cases and so this procedure was discontinued.

Subjective changes, weight, frequency of cough, and amount and color of expectoration were recorded at each subsequent visit.

OBSERVATIONS.

1. Chronic Mucopurulent Bronchitis.—The twenty patients with chronic bronchitis of the mucopurulent type ranged from 16 to 70 years of age. None worked in the dusty trades. Two had peripheral arteriosclerosis, four had hypertension and peripheral arteriosclerosis, and two had emphysema. One patient had tabes dorsalis, and another had asymptomatic tertiary syphilis and chronic fibroid phthisis with negative sputum. There was no evidence of myocardial failure in any of these cases.

There was absence of history of frequently recurring sore throats, colds or other upper respiratory infections in only two cases. Five patients were subject to frequent colds with coryza, rhinitis and sore throat. Two patients had had occasional sore throats with rhinitis and acute sinusitis. Nine patients had had an occasional sore throat or an infrequent mild cold. One patient told of having had four attacks of pneumonia during childhood with subsequent periods of postnasal discharge and foul breath, and another patient remembered one attack of pneumonia twenty years previously, with only an occasional sore throat since that time. One patient had had influenza in 1918, but the symptoms of chronic bronchitis were first noticed about eight years later.

Thirteen cases showed evidence of disease in the nose, throat or sinuses, such as hypertrophy of the mucous membrane, deviation of the septum, sinusitis or postnasal discharge. The duration of the cough varied generally from less than a year to fifteen years, but exceeded twenty years in four cases. The symptoms were worse at night in six cases, on arising in the morning in

seven, and were about equal at night and in the morning in the remaining seven cases.

The amount of sputum varied during a twenty-four hour period from one teaspoonful to a cupful or more. In seven cases there was more than a quarter cupful.

Roentgenogram of the thorax before bronchoscopy was negative in fifteen cases, and in five cases roentgenologic data were as follows: In three cases increased markings at the bases, suggestive of bronchiectasis; in one case, cicatricial changes at the left base and displacement of the heart to the left without evidence of bronchiectasis, and in one case, an oval dense shadow in the right lower lobe which suggested a malignant new growth.

On bronchoscopic examination a normal bronchial mucous membrane was observed in four cases, only the slightest abnormalities in three, and swelling or thinning of the bronchial mucous membrane in the remaining thirteen. In addition, variable amounts of tenacious mucopurulent secretion, inflammation, ulceration or stenosis were found in several cases. A swollen mucous membrane with increased amount of secretion was present in seven cases. In two cases an injected mucous membrane was the only abnormality found, and in two other cases the bronchial mucous membrane was thin and glistening. Hypertrophy, atrophy, inflammation and secretion were observed in one case.

After instillation of lipiodol, further information was obtained from roentgenograms of the lungs in five cases. In the first case unsuspected diffuse bronchiectasis at the base of the right lung was revealed. A suspicion of bronchiectasis in the second case was dispelled by roentgenogram after instillation of lipiodol. The third case, which had a normal appearing roentgenogram, now revealed marked bronchiectasis at the left base which apparently had been hidden by the shadow of the heart. In the fourth case a suspected malignant tumor proved to be a sharply circumscribed encapsulated fibrosed abscess. In the fifth case suspected bronchiectasis was made perfectly evident with lipiodol.

After introduction of the bronchoscope and aspiration of secretion, followed by instillation of lipiodol, the most striking observation was the relief experienced by the patients. Usually the improvement began within two weeks and gradually progressed.

Complete disappearance of cough and expectoration with subjective improvement and gain in weight occurred in eight cases; the patients were so greatly improved that they were considered clinical cures. Marked decrease in symptoms with subjective improvement and freedom from colds followed in nine cases. One patient reported no diminution in the frequency of colds, but was free of cough and expectoration between such attacks. Two patients did not show improvement. One patient with severe bronchiectasis had several aspirations and improved steadily after each one until his symptoms were practically absent.

2. Chronic Bronchitis with Bronchial Asthma.—There were three cases of chronic bronchitis with bronchial asthma. There was no demonstrable nasal, pharyngeal or sinus disease at the time of examination. Each of the patients gave a history of frequent colds and sore throats, and two patients had had sinusitis. The symptoms dated back five, fifteen and ten years, respectively.

Bronchoscopic examination revealed a pale mucous membrane with a moderate amount of clear, very tenacious secretion in all three cases and the roentgenogram of the thorax was negative before and after instillation of lipiodol.

After bronchoscopy, aspiration and instillation of lipiodol, one patient reported disappearance of cough and expectoration, and complete freedom from asthmatic attacks for one year. She subsequently moved to another city and we were unable to communicate with her. The second patient did not derive benefit from the procedure. The third patient reported great benefit after the first bronchoscopy, and returned in two weeks asking for another treatment. Progress notes over a period of eighteen months following the second treatment showed no recurrence of asthmatic attacks.

Since this study was completed, an additional case of a similar nature has come under observation; the patient has received treatment, but the benefit has not been striking.

3. Chronic Cough Without Expectoration.—Two cases of chronic cough without sputum were studied in a similar manner. Clinically they were classified as catarrhe sec of Laennec, but subsequent study with the aid of the bronchoscope disclosed that

the symptoms were due to bronchostenosis. In one case the narrowed portion of the bronchus was dilated and prompt improvement followed. In the other case dilatation was performed and subsequent improvement did not occur.

SUMMARY AND CONCLUSIONS.

1. Twenty-five cases with chronic cough of unexplained origin were studied with the aid of the bronchoscope. Aspiration of visible secretion followed by injection of lipiodol was carried out in each case. Twenty of these patients had chronic productive cough, three had productive cough associated with bronchial asthma, and the remaining two had chronic cough without sputum.

2. Various changes were visible through the bronchoscope in twenty-one of the twenty-five cases. The changes noted were swelling and inflammation of the mucous membrane, thinning of the mucous membrane, abnormal secretion or stenosis.

3. In contrast to the observations of others,[1 2 3 4] our observations do not support the view that such changes in the bronchi are always secondary to disease in the nose, throat or sinuses.

4. The belief that chronic bronchitis is due to the presence of undetected small bronchial dilatations[5] does not coincide with our observations.

5. Instillation of lipiodol was helpful in the recognition of obscure bronchiectasis, and possibly of value in the treatment of chronic bronchitis. The beneficial therapeutic effect of lipiodol has been observed by others,[5 6 7 8] and the value of aspiration alone has also been recognized.[4 9 10 11 12]

6. After bronchoscopic aspiration and instillation of lipiodol, improvement, as manifested by disappearance of cough and of expectoration, with gain in weight and a sense of well-being, was observed in ten cases. Less improvement was observed in ten cases, and no improvement in five cases.*

25 BENNET ST.

*We wish to thank Dr. H. J. Inglis, Chief of the Otolaryngological Clinic, and Dr. J. J. Pratt, Chief of the Medical Clinic, The Boston Dispensary, who have so generously placed the material for this study at our disposal, and for other suggestions which they have made during its progress.

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LXXXIX.

NONOPAQUE FOREIGN BODIES IN THE FOOD AND AIR PASSAGES.*

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In the case of a sick child the possibility of a foreign body in the food or air passages should always be considered. It is surprising how many times a foreign body is discovered to be the cause of a protracted illness which might have been greatly shortened by early removal of the intruder. A sense of false security is often engendered by the fact that a symptomless interval very often follows after the initial distress from a foreign body has subsided. The family physician is, of course, first consulted in nearly all cases, and upon him rests the responsibility of being informed as to the symptoms which make it possible to diagnose the presence of a foreign body. That this responsibility is being well met is demonstrated by the fact that in one large clinic about 90 per cent of cases in which foreign bodies were found had been correctly diagnosed by the physicians who sent in the patients.

When a foreign body is inhaled a person usually suffers a violent choking spell, and both he and his associates are often thrown into a panic. In this state of mind parents often inflict serious injury to a child's throat by attempts to remove a foreign body with the fingers. Such old methods as trying to remove a foreign body from the esophagus with a coin catcher or bristle probang, etc., are obsolete, because these instruments are capable of inflicting serious damage, even to the extent of perforating or rupturing the esophagus. Neither should an attempt be made blindly to push the foreign body into the stomach, because of the same possibility of producing fatal injury.

When an obstructive foreign body is inspired into the larynx and becomes impacted there, death is very likely to ensue. A patient can die in the unbelievably short time of two minutes if

*Read before the Panhandle District Medical Society, Amarillo, Texas, April 11, 1933.

he is struggling against a blocked airway. The bad practice of inverting a patient by the heels and pounding him on the back for the purpose of dislodging a tracheal foreign body may impact it between the vocal cords and precipitate death by suffocation.

The history, physical examination, and proper interpretation of the physical findings will often lead to a suspicion of a foreign body. A positive history of a choking spell may be valuable, but a negative history as regards choking should be considered valueless. The patient may be too young to report such an experience, or the foreign body may have entered practically unnoticed. If there has been a choking spell, followed by hoarseness, aphonia, dyspnea, cyanosis or dysphagia, this is a most important lead which should be elaborated as much as possible. It is important to discover what the patient had in the mouth prior to the attack, and a duplicate of the suspected foreign body is of the utmost assistance to the endoscopist in planning a solution of the mechanical problem involved in its removal. The X-ray is often essential in clearing up doubtful findings. Even after all findings are recorded and considered, the determination of the presence of a foreign body may rest upon a diagnostic bronchoscopy or esophagoscopy.

Dr. Chevalier Jackson makes mention of three characteristic signs of foreign body in the air passages. He describes an "asthmatoid wheeze," which is best heard by placing the ear or the bell of the stethoscope at the open mouth of the patient during breathing. When the foreign body is loose in the trachea, an "audible slap" is often heard as a result of its being coughed or flung against the glottis during expiration. The same shifting of the foreign body may cause a "palpable thud" which can be felt with the finger placed upon the larynx.

Two other important symptoms are mentioned by McCree, namely, a tracheal flutter, and a limitation of expansion on the affected side.

Physical findings may detect an obstructive emphysema on the affected side, due to a check valve action of the foreign body. During the inspiratory cycle the widening of the bronchus allows air to enter the lung alongside the foreign body. During the expiratory cycle the narrowing of the bronchus retards or pre-

vents escape of the air, and by this process the portion of the lung beyond the foreign body is pumped up, resulting in the emphysema. The area thus affected will give back a hyperresonant percussion note. The same overdistension will cause limitation of expansion on this side, and will cause the diaphragm on the affected side to be lower than normal, especially on expiration. The heart will also be displaced away from the emphysema. There will be diminished intensity of breath sounds distal to the foreign body. If the foreign body completely blocks a bronchus, there will result an atelectasis or massive collapse, and, later, a drowned lung. Vocal resonance and vocal fremitus are lost when this ensues, and an erroneous diagnosis of empyema might be reached.

There is a great tendency for foreign bodies of vegetal origin to cause excess bronchial mucus and a patient may drown in his own secretion. The removal of this secretion by a suction tube is of the utmost assistance and may prevent this tragic outcome. Uncooked dry beans are probably the most dangerous of all foreign bodies in the air passages, beause the warmth and moisture in the lungs causes a great enlargement by swelling, and a violent laryngotracheobronchitis develops from some unknown quality of vegetal substances. Peanuts likewise exert a most malign influence, and watermelon seeds are not much better. Grass burs cause a most intense inflammatory reaction, probably from the fact that their spines penetrate and infect the mucosa.

There are numbers of cases on record with multiple foreign bodies, and I have had numerous instances of this in my own experience. The removal of a foreign body should not satisfy the operator, but he should reintroduce his instruments and assure himself that nothing foreign is visible below the site of the foreign body already removed. After an interval of two or three days another X-ray examination should be made to demonstrate that the esophagus is unobstructed or that the lung pathology has cleared up.

It is most unfortunate when a foreign body in the lungs is not recognized, for measures which are not indicated may be instituted for relief of lung findings which are misinterpreted. Gabriel Tucker relates the instance of one patient on whom a surgeon

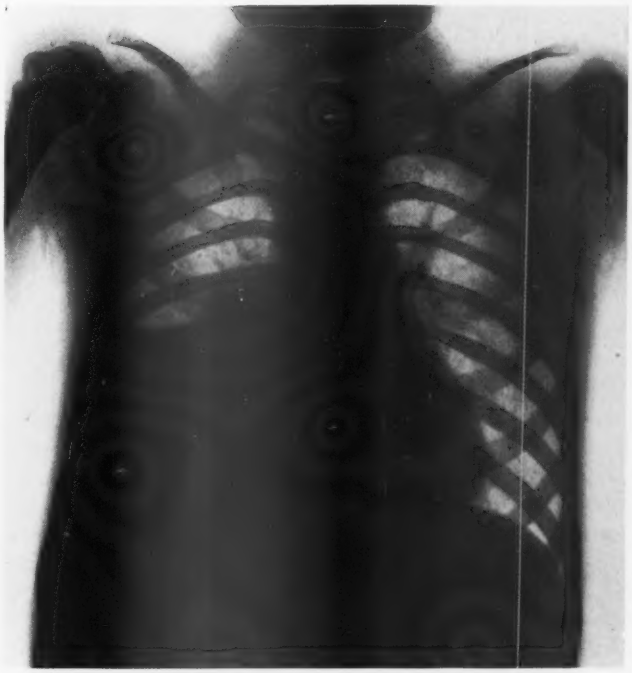

Fig. 1. Nonopaque foreign body, pecan kernel, in lower right main bronchus for four days in boy of seven years. While the foreign body cannot be visualized, there is an atelectasis of the right middle and lower lobes due to complete obstruction of bronchus below point where upper lobe bronchus is given off. Prompt recovery after bronchoscopic removal.

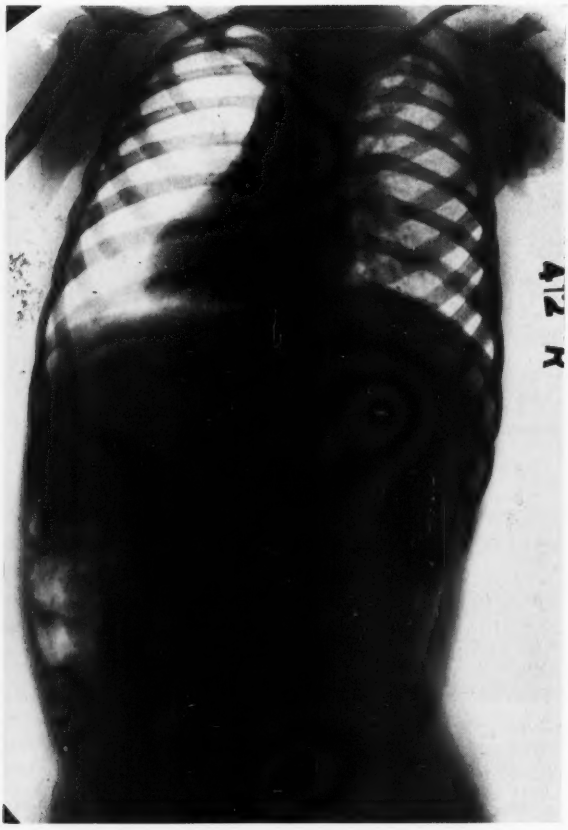

Fig. 2. Obstructive emphysema of left lung, due to fragmented corn in left main bronchus. Prompt recovery after bronchoscopic removal.

performed a thoracotomy for suspected "empyema" which did not exist, which of course brought negative results. The patient was subsequently cured by the bronchoscopic removal of a foreign body from the lungs.

When there is a partial obstruction to the entrance of air into the lungs, the voluntary muscles of the chest are brought into play to assist in getting the air past the obstruction. For this reason a general anesthetic is very dangerous in dyspneic patients, because in a state of narcosis the voluntary muscles are thrown out of function and the margin of safety is enormously reduced. Moreover, children do not stand local anesthetics well; and for these reasons no anesthetic, neither general nor local, is used in children when a good endoscopic team is working together, and only a local anesthetic or none at all in adults. Suitable methods of restraint are available which make it possible to control a child's struggles and pass the instruments without pain.

In the case of a foreign body in the esophagus there is generally some dysphagia and occasionally an aphagia. When nothing will pass through the esophagus, there is need for hasty relief in order to prevent death by thirst or starvation. It is rather common to find the esophagus occluded by food lodged above an esophageal stricture from lye burns, and I have seen little patients reduced to the most extreme emaciation from this cause. Happily, it is often possible to dilate these strictures under direct endoscopic guidance so that the patients can swallow normally and keep fully nourished. Due to its sharp ends or sides, or from constant pressure, a foreign body in the esophagus may perforate or ulcerate through the esophageal wall into the trachea or mediastinum. We have had one case where a mass of pork became impacted in the esophagus and caused a pressure necrosis of the esophagotracheal wall, so that a fistula into the trachea developed, through which food and fluids passed into the lungs and produced a lung abscess. Rupture of the esophagus into the mediastinum generally results fatally as a result of the infectious process which develops.

The constant pressure of a shoe last on the ensiform process of an old shoemaker caused such a depression that it partially obstructed the esophagus, and at this point an enormous mass of

turkey meat became impacted. This patient was in extreme distress from hunger and thirst and pressure of the foreign body until it was removed by esophagoscopy.

One of the objective symptoms of foreign body in the esophagus is the finding of secretion in the pyriform fossæ, which lie behind and lateral to the larynx. Another suggestive sign is pain opposite to the side on which pressure is applied to dislocate the larynx laterally. A child with a foreign body in the esophagus is very prone to assume a characteristic posture, namely, face downward on the bed, as this seems to reduce its discomfort.

The X-ray will graphically reveal the presence of a radiopaque foreign body in either the food or air passages. In the case of a nonopaque foreign body the problem is more difficult. The lung changes produced by either type of foreign body are much the same, except that the inflammatory reaction is much greater in the case of vegetal substances, while inert substances are often tolerated with remarkably little immediate reaction. It is not necessary, therefore, actually to visualize the foreign body; but an understanding of the lung changes produced by a foreign body will reconcile the physical findings and the X-ray findings.

A nonopaque foreign body in the esophagus can sometimes be localized by having the patient swallow a capsule filled with some opaque medium such as lipiodol or barium or bismuth. The foreign body will often arrest the descent of the capsule and thus reveal its location with the fluoroscope or X-ray film.

That the nonopaque foreign body is a frequent intruder is borne out by the fact that in our collection of 137 foreign bodies removed from the food and air passages, 62, which is practically 47 per cent of the total, have been nonopaque from the standpoint of the X-ray. However, a large number of these have produced characteristic changes which the X-ray can demonstrate.

It has been estimated that more than 90 per cent of foreign bodies in the food or air passages can be successfully removed without damage to the patient by bronchoscopy or esophagoscopy.

SUMMARY.

1. In a sick child always think of the possibility of a foreign body in the food or air passages.

2. Death by suffocation may follow attempt to expel a foreign body by pounding the patient on the back.

3. Attempted removal of a foreign body with the fingers or obsolete instruments may inflict serious or fatal injury.

4. Physical signs produced by foreign bodies are rather characteristic.

5. The X-ray can localize a foreign body in the lungs or esophagus without actual visualization of the foreign body, which may be nonopaque.

6. Skillful endoscopic measures will successfully and harmlessly remove more than 90 per cent of foreign bodies lodged in the food or air passages.

7. Possibly a positive history of a choking spell receives less weight than it deserves. Sometimes physical signs and X-ray findings are all negative, but a positive history will lead to bronchoscopy or esophagoscopy and removal of a nonopaque foreign body.

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XC.

SIGNIFICANT ANATOMIC FEATURES OF THE AUDITORY MECHANISM WITH SPECIAL REFERENCE TO THE LATE FETUS.*

DOROTHY WOLFF, PH. D.,

ST. LOUIS.

I. INTRODUCTION.
- A. HISTORICAL REVIEW.
- B. ANATOMIC MATERIAL.
- C. METHODS.
  1. Autopsy Technic.
  2. Preparation of Histologic Specimens.
  3. Preparation of Wax Reconstruction Based upon Photographic Negatives.
  4. Description of the Model.

II. OBSERVATIONS.
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  2. Shape.
  3. Walls.
  4. Contents.
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     b. Muscles.
     c. Pyramidal Eminence.
     d. Sinus Tympani.
     e. Nervus Facialis.
     f. Auditory Tube.
     g. Mesenchyme.
     h. Membrana Tympani.

I. INTRODUCTION.

A. HISTORICAL REVIEW.

Politzer in his "Geschichte der Ohrenheilkunde," Vol. I, 1907, indexes 203 names of important contributors to the history of otology from the time of the Greeks to the middle of the nineteenth century. In Volume II he lists about 850 names from the

*From the Department of Anatomy, Washington University School of Medicine, with the aid of the Science Research Fund granted by the Rockefeller Foundation.

middle of the nineteenth century to 1911. From these imposing figures it is obvious that only a few of the most important steps in the development of our knowledge of the anatomy of the ear can be suggested in the present paper.

Ball states that the cochlea of the ear was named by Empedocles (490-430 B. C.). The Greeks recognized the presence of the auditory tube and believed the cavity of the ear was filled with air. It is to Galen (131 B. C.-201 A. D.) that we owe the accurately descriptive term "labyrinth." Galen followed the course of the facial nerve and described the mastoid and zygomatic processes. To the drum membrane he ascribed but little importance.

According to A. Wilder and to Ball, Allesander Achillini of Bologna (1463-1512) first described the malleus and incus. Politzer believes that these ossicles were known prior to this time. Ingrassius (1510-1580) discovered the stapes in 1548. He also discovered the tensor tympani muscle, an observation sometimes attributed to Eustachius.

Three contemporaries, all great men of their time, contributed notably to otologic investigations. These were Eustachius (1510-1574), Vesalius (1514-1564), and Fallopius (1523-1562). Eustachius was not the first to observe the tube which bears his name, but he described it accurately and realized its significance. According to Siebenmann, he divided the acoustic nerve into three portions. Eustachius' illustrations show that he was familiar with the facial canal, the oval and round windows, the carotid canal, antrum and even the endolymphatic duct.

Vesalius was the first to publish illustrations of the middle ear and ossicles (1543) but he failed to observe the stapes.

Of the many valuable contributions to anatomy made by Fallopius, his discovery of the trigeminal, auditory and glossopharyngeal nerves most concerns us. He saw the chorda tympani but did not recognize its significance. Politzer states that he made a careful study of the hearing organs of different ages, observing that the mastoid process was absent in newborn and gradually increases in size with the process of growth. Siebenmann tells us that he named the fenestra rotunda and the fenestra ovalis.

Du Verney (1648-1730), in his "Tractatus de Organo Auditis," gives the first accurate description of the cochlea and an interpretation of its function. On page 23 of this volume we find:

. . . Cum enim latior fit, ad initium primi gyri sui, quam ad finem ultimi, ubi in acuminatam desinit figuram, adeoque caeterae eius partes ratione latitudinis paulatim diminuantur, dici potest, partes eius latiores moveri posse immotis caeteris, adeoque tremere tantum lentius, quod gravibus tonis convenit; e contrario, cum partes eiusdem angustiores impelluntur, quorum tremores citiores sunt, atque ita tonis acutis respondent, perinde ut fieri solet in chalybe in spiram contorto, cuius partes latiores tardius tremunt, & tonis gravibus respondent; partes ve o minus latae, & frequentius & velocius, adeoq; cum tonis acutis conveniunt: ita tandem secundum diversos laminae spiralis motus, spiritus nervi, in substantiam eius divisi, diversas recipiunt impressiones, quae cerebro varias tonorum figuras repraesentant.

. . . Since it is broader at the beginning of the first gyrus than at the end of the last, where it terminates in a pointed design, and the remaining parts gradually diminish in diameter, it can be said that the broader parts can be moved while the rest are unmoved and so vibrate more slowly as is suitable to the lower tones. On the other hand when the narrower parts are struck, the vibrations of which are swifter and so correspond to the higher tones, just as in the case of a trumpet curved into a spiral whose wider parts vibrate more slowly and so are suited to the lower tones, but the less wide parts vibrate more swiftly and so are suited to the higher tones, so according to the different motions of the spiral lamina, the vital elements ("spirits") of the nerve divided throughout its substance receive different impressions which represent different tone designs to the cerebrum.

Here we have the same idea which Corti developed at a later period. Corti's statements were, of course, based upon microscopic observations of the interior of the cochlea. We now believe the basal turn receives the higher tones instead of the lower. Corti's observations explain the seeming discrepancy.

Valsalva (1666-1723), in his "De Aure Humana Tractatus," instituted the terms scala vestibuli and scala tympani in preference to scala superior and scala inferior. He believed the lamina basilaris to be a septum of musical bands tense and resonating, whose larger width was at the base of the cochlea rather than at the apex. Thus we have a further development of the resonance theory, although the localization of high tone reception, like du Verney's, is not that generally held at the present time. Valsalva describes three muscles attached to the malleus.

Politzer says Cotugno was the first who had the courage to break the old tradition that the labyrinth contained air and not fluid. Corti tells us that Theodor Meckel (son of the famous Johann Friedrich Meckel), in 1777 proved that liquid and not air was contained in the labyrinth. But since Cotugno's paper appeared in 1760 he receives priority in the matter. Cotugno described the aqueducts of the cochlea and vestibule, and knew that small veins accompany these. He discovered the endolymphatic fluid and claimed that there were two circulations in the labyrinth, a greater and a lesser. He explained the whole theory of resonance, the response of a single chord to a single tone, the localization of low and high tones, etc. (Gradenigo.)

Alphonse Corti (1851), in his "Recherches sur l'organe de l'ouie des mammiferes," presents the first detailed description of the sensory end-organ of hearing. Accurate measurements of the membranous and bony spiral laminæ are given. The observation made for the first time by du Verney (although apparently unknown to Corti), that the bony spiral lamina in the basal turn is broader than in the apical turn, was further established by Corti's observation that the membranous spiral lamina in the basal turn is shorter than in the apical. (This is a most significant observation when related to the fact that low tones are received in the apical turn and high tones in the basal turn of the cochlea.) The author divided the organ into the pectinate zone and the dentate zone. He observed the pillar cells and the different ranks of "toothed cells," now known as hair cells, also the tectorium. He noted that the perivascular spaces were three times larger than the vessels in them. He refuted Kolliker's idea that there was a muscularis cochlearis. He observed pigment cells and considered them to indicate a pathologic storing of hematin.

Scarpa (1747-1832), according to Rezius, first described the existence of a similar membranous labyrinth in numerous forms from fish to higher vertebrates. He described the two membranous vesicles in the vestibule and the spiral course of the cochlear nerve as it proceeds through the sievelike modiolus. Routine examination of autopsy material was apparently inaugurated by Toynbee in 1852 and 1853. He gives the case histories, findings

(primarily gross), and the method for procuring the temporal bones at autopsy.

Enumeration of names already familiar to the reader is but dull tribute to the honor of the many modern investigators who have increased our knowledge concerning this complicated organ. Rezius' monumental work is evidence of the wealth of time and labor put upon the study of the auditory mechanism in the latter part of the nineteenth century.

B. ANATOMIC MATERIAL.

The following pages are devoted primarily to a description of conditions found in the petrous portion of the temporal bones of a six-months, premature, negro infant (No. 3887). A wax reconstruction of the right otic capsule of this specimen was made to elucidate certain points. In addition to the particular specimen described in detail, reference is freely made to microscopic observations of sixty pairs of human temporal bones. This material ranges in age from late embryonic life to 70 years. Among the specimens younger than 6 fetal months are included one embryo of less than 2 months, one of 2¾ months, one fetus of 3 months (whole head sectioned), one of the fourth month (half head sectioned). This latter specimen was lent through the kindness of Dr. L. Guggenheim. The microscopic study was reinforced by observations from gross dissections of four fetuses estimated to be approximately 6 months. Certain animal material has been studied in comparison with the human specimens: monkey (Pithecus), dog, albino rat, and cat. Postnatal human material has been used for the study of the amount of mesenchyme in the tympanum at given ages, the enclosure of the facial nerve by its bony canal, and the patency of the subarcuate fossa.

Abstracts from the case history of No. 3887 follow:

A male negro infant was delivered outside the hospital in the sixth month of fetal life. Delivery was spontaneous and duration of labor was seventeen hours. The infant was transferred immediately to the hospital for incubation. The nose and ears were negative. The child was kept alive in the hospital by giving oxygen and CO_2 as a respiratory stimulant, but the respiration gradually became weaker and finally ceased seven hours after birth.

The following data are taken from the autopsy protocol:

The body measured 32 cm. in length and weighed 600 gm. All tissues appeared normal for the premature infant of six months' gestation except for a hemorrhagic condition. This existed in the subcutaneous tissue, in the pancreas, and in the meninges. Excessive meningeal hemorrhage extended over both cerebral hemispheres. A diagnosis of syphilis could not be made from the gross and microscopic findings in these tissues.

The following report is taken from the author's personal observation:

Nasal Sinuses.—In one area the ciliated epithelium was found to be sloughed off. Free red blood cells and a few leucocytes were found spread along the surface of the epithelium as if caught in the cilia. Congestion of the subepithelial vessels was marked. Occasional mononuclear eosinophilic myeloblasts were seen scattered in the subepithelial tissue.

Temporal Bone Sections.—Frank pus occurred in the lumen of the middle-ear cavity on each side. Very slight infiltration of the mesenchyme had occurred, however. Petechial hemorrhages were present in the subepithelial tissue. The capillaries of the subepithelial tissue and the mesenchyme were greatly congested. Malformations of the bony contour of the superior and horizontal canals existed. Marked evidences of prematurity were present which will be discussed in detail under the anatomic discussion.

C. METHODS.

Autopsy Technic.—The method of procuring and preparing the material is essentially that described by Eckert-Möbius, and that first employed on a large scale in this country by Dr. S. J. Crowe of Johns Hopkins University Medical School.

The material from No. 3887 was obtained twenty-four hours after death. Fig. 1 indicates that the membranous labyrinth is in fairly good state of preservation in spite of this fact. Postmortem changes had occurred, but they are readily differentiated from the pathologic processes during life. (The ideal time is two hours after death.) This specimen was so immature that the material was removed from the cadaver with strong scissors. When older or adult material was obtained the technic was as follows: The autopsy was begun on the head first whenever that was possible, in order that the delicate membranous parts of the ear might be put into fixative before dissolution had set in.

The brain was first removed. (In approaching the region of the ear it is advisable not to strip the dura from the petrous bone, as in so doing the endolymphatic sac will be torn off.) The petrous portion of the temporal bone was removed in the following manner: An electric circular saw was inserted at the apex of the petrous part and that portion of the bone was cut through.

The saw was then removed and inserted at the base of the petrous portion. When this part had been well cut the saw was again removed and inserted so as cut through the floor of the skull at about the anterior margin of the pyramid, in a lateromedial direction parallel to the superior angle of the petrous bone. This cut was planned and made so as to include behind it the whole middle ear cavity and tympanic membrane. (In this connection it is well to remember that the tympanic membranes of the young infant and fetus lie in a more horizontal plane than do those of older specimens.) The final sawcut was made by inserting the saw on the floor of the posterior cranial fossa and following the posterior angle of the petrous bone. When the bony parts had been sawed through, the soft parts were cut with scissors. A chisel and hammer may be used instead of the circular saw. It may be said, in passing, that the problem of procuring a portion of the Gasserian ganglion and the petrous apex at autopsy is not a difficult one and the material secured is of much interest for microscopic investigation. The prosector needs merely to direct his initial cut far enough medially and anteriorly to include the ganglion.

When the desired blocks of the otic region had been removed the defects of the skull were closed with plaster of Paris and the skull cap replaced. The tissue-blocks were tagged as to autopsy number, right and left side, and plunged into Zenker's solution, or 10 per cent formalin, immediately after their removal from the cadaver.

Preparation of Histologic Specimens.—From this point on the exact technic for specimen No. 3887 will be given. The Zenker's solution used contained no acetic acid. The following schedule was adhered to:

Zenker's solution	24 hours
Washed in running tap water	22 hours*
Alcohol, 70 per cent	7 hours
Alcohol, 95 per cent	21 hours

At this point the blocks were suspended in a wide-mouthed, brown glass bottle half filled with an aqueous 5 per cent solution of nitric acid. It was observed that gas bubbles formed quickly

*According to S. R. Guild's results, it is now considered better technic to use degassed water for all purposes.

on the surface of the block after it was plunged into the acid. Frequent agitation caused these bubbles to rise, thus doing away with the gas film that would otherwise have formed around the block, permitting the nitric acid to come more directly in contact with the tissues.

Fresh HNO_3	16 hours
Fresh HNO_3	18 hours
Washing in running tap water	16 hours
5 per cent Na_2SO_4	24 hours
80 per cent alcohol with iodin	2 days

Drops of saturated solution of iodin were added to the 80 per cent alcohol until a champagne color was obtained. As this bleached, more iodin was added until the bleaching no longer occurred.

80 per cent alcohol without iodin	24 hours
90 per cent alcohol	24 hours
100 per cent alcohol	24 hours
Ether alcohol	24 hours
Thin celloidin	5 days
Thicker celloidin	9 days
10 per cent	10 days
15 per cent	10 days

Each block was then cut serially at 20 micra, stained in Harris' hematoxylin and eosin and mounted in neutral balsam. Following Dr. E. V. Cowdry's suggestion, cedar oil has since been found more satisfactory for mounting, as there is no discoloration of the medium after years of service. A few sections were stained in Mallory's connective tissue stain and in Giemsa's stain.

Preparation of Wax Reconstruction Based Upon Photographic Negatives.*—A negative model of the right ear of specimen No. 3887 was constructed for further study. A series of photographic negatives was taken at a magnification of 25 times. This was done by projecting the image of the microscopic section on sensitized paper in a dark room, according to the method of Dr. W. H. Lewis and Dr. G. L. Streeter. A Leitz projection motion picture apparatus was used. The film holder was removed and two slide clips attached in its place. The paper found most satisfactory was Eastman's smooth contrast bromide P. M. C., size 11 by 14 inches.

In choosing which sections were to be photographed, the best in each successive group of three was taken. Exposures were

*Bourne method modified.

almost instantaneous, being just as long as it took to turn a snap-switch or count "one-and." Each sensitive paper was marked with a soft lead pencil with the number of the specimen and of the section, as No. 3887 rt., sec. 304. When the paper is exposed these marks will prevent the light from penetrating and the notation will then appear on the finished negative as white figures on the black background, as shown in the upper left corner of Fig. 1.

The negatives were developed in the regular elon-hydroquinone solution (Eastman formula D-72), rinsed in "acid short stop" (formula SB-1), and fixed in "acid hardening fixing bath" (formula F-1). The prints were washed in running water several hours, rolled on plate glass with a squeegee roller; next placed in a tray containing 10 per cent glycerin, remaining one-half hour, and again rolled on the glass. They were then spread over a stretcher of cheesecloth, covered by sheets of heavy white blotting paper. When dry the prints were stored in the black envelopes in which the sensitized paper originally came.

As has been stated, the sections were cut at 20 micra. Thus each section was 20/1000 or 1/50 mm. thick. By using a magnification of 25 times, each section was estimated to be proportionately 25/50 or ½ mm. thick. Three sections were calculated to be 1.5 mm. thick. Therefore, by photographing one section in every group of three, it was possible to use wax plates of 1.5 mm. thickness. This thickness is most advantageous for handling and cutting large sheets of wax.

The method of pouring the wax was essentially that described by Guyer. The wax formula used was 90 grams of beeswax, 30 grams of paraffin, 30 grams of cornauba, and 30 grams of white lump resin. This formula proved highly satisfactory for the very hot summer temperature of St. Louis. But during the cold weather the wax was much too brittle for safe and accurate cutting. It is not recommended. The wax was cut in sheets 11x14 inches, and upon these was traced the desired outline from the photographs. An engraver's tool, No. 3, sharpened to a rounded point, was used for tracing. For cutting, a similar tool, sharpened like a two-edged sword, was used.

One hundred and twenty-two sections were photographed and traced on the wax sheets. This gave a record in three dimensions of the human ear from the upper level of the endolymphatic sac and just below the level of the arch of the superior semicircular canal, down to the lower level of the promontory and niche of the round window. This model was constructed so as to show the relationship of the endolymphatic and perilymphatic systems. The perilymphatic spaces were cut out of the stencil, leaving the bone solid and the endolymphatic system solid except for that part of it which possesses no perilymphatic spaces—i. e., the endolymphatic duct and sac. These were cut out. The model was then painted with duco, the bony parts in French gray, the perilymphatic walls in buff, the endolymphatic parts in ivory and the nervous elements in yellow. In certain places, where it was found to be too arduous a task to hold small nerves in place, their canal walls were merely painted yellow.

Description of the Model.—The model was then made dissectible at seven levels. This gave nine sections, or "blocks," as they have been designated, in order not to confuse the reader with microscopic "sections." For convenience in piling they were numbered from the bottom upward. A wooden frame supporting glass plates was constructed in sections to support each block of the model. This makes a satisfactory means of preventing sagging of the wax in hot weather.

Clarity in presentation is gained by discussing the blocks in order reversed from the way in which they are piled—that is, the way the microtome proceeds in cutting the material. Making the model dissectible at various levels has the advantage of the possibility of making comparisons with the actual microscopic section of a given level and is hence invaluable for teaching purposes. Furthermore, the different blocks may be inverted and thus studied from below. Photographs of each block were taken.

The levels chosen for dissection were as follows:

Block 9 (Sections 127-159, Fig. 2) shows the saccus endolymphaticus in the dura, the peripheral position of the endolymphatic system in the bony canals, the internal acoustic meatus with the nerves and the cochlear artery. The block begins at a level just below the arch of the superior semicircular canal and

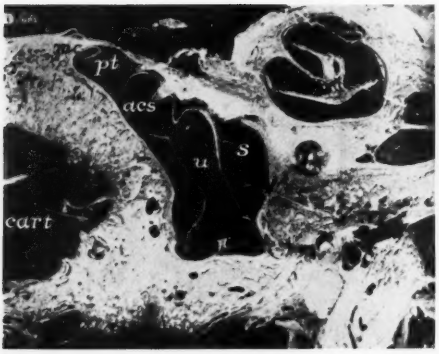

Fig. 1. Negative of Sec. 304, Specimen No. 3887. Reduced from one of the originals from which the wax plates were traced: *acs*, ampulla canalis superioris; *cart*, cartilage; *mc*, membranous canal; *pt*, perilymphatic tissue; *s*, saccule; *u*, utricle.

cuts into the basal turn of the cochlea. At this level we get an inkling of the petrous bone in the process of development, for we find the internal acoustic meatus appearing high on the posterior wall.

Block 8 (Sections 161-203, Fig. 3). Here we have a good presentation of the expansion of the membranous superior canal as it approaches the crus commune, also of the irregular contour of the cavity of the endolymphatic sac. The internal acoustic meatus is shown, within which is seen the mass of the facial and acoustic nerves. In the posterior half of the block appears the large opening of the subarcuate fossa. The reverse side of this block (Fig. 4) depicts clearly the direct course of the vessel ac-

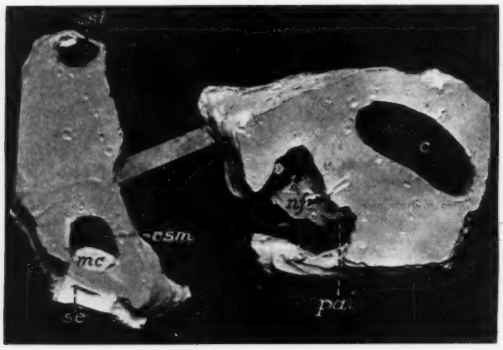

Fig. 4. Superior level of model. The incomplete formation of the bony capsule is evidenced by the separation of the cochlear and vestibular elements: *c*, cochlea; *csl*, lateral limb of can. sup. semic; *csm*, medial limb, ibid.; *nf*, nervus facialis; *pai*, porus acusticus internus; *se*, saccus endolymphaticus.

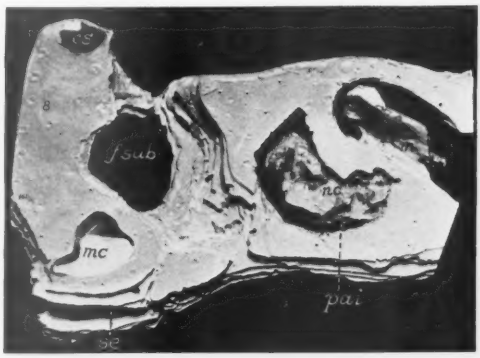

Fig. 3. Block 8, Sec. 161. Showing the medial limb of the canalis semicircularis superior entering the crus commune. Saccus endolymphaticus is well seen here: *f sub*, fossa subarcuata; *m*, modiolus.

companying the endolymphatic duct into the crus commune. The endolymphatic duct having gained the anterior border of the crus commune follows it for some distance. During this part of its course it meets a limb from the utriculo-saccular duct. Beyond this point it enters the crus commune but maintains the identity of its membranous canal. In this block also may be studied the radiate distribution of the cochlear nerve. The modiolus, cochlear duct and scalæ are also shown.

In Block 7 (Sections 205-233, Fig. 5) the advance of the facial nerve toward the tympanum is seen. The location of some of the foramina in the fundus of the internal acoustic meatus can be studied. Here again may be observed the radiate distribution of

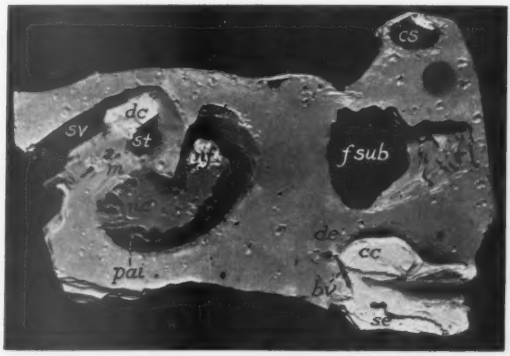

Fig. 4. Block 8. Inferior view showing ductus endolymphaticus approaching crus commune, *cc*; *bv*, blood vessel *dc*, ductus cochlearis; *nc*, nervus cochlearis; *st*, scala tympani; *sv*, scala vestibuli.

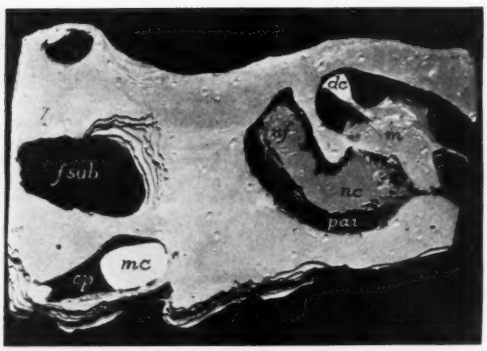

Fig. 5. Block 7, Sec. 205. Nervus facialis is seen approaching the cavum tympani. The 7th and 8th nerves have not been differentiated from each other in the nerve mass in the internal meatus in modeling: *cp*, canalis semicirc. posterior.

the cochlear nerve. The subarcuate fossa appears as a great space between the limbs of the superior semicircular canal.

Block 6 (Sections 235-259, Fig. 6) shows the approximate termination of the subarcuate fossa. One branch of the cochlear nerve is seen diverging to supply the prolonged basal turn which is not included in the model. The ramus posterior of the vestibular nerve diverges at this point to supply the posterior ampulla (Fig. 6, *rp*). Nervus facialis is seen leaving the confines of the otic capsule and proceeding laterally. The ductus endolymphaticus has not yet entered the sacculus.

Block 5 (Sections 264-300, Fig. 7). The superior branch of the vestibular nerve is here seen approaching the ampullæ of the

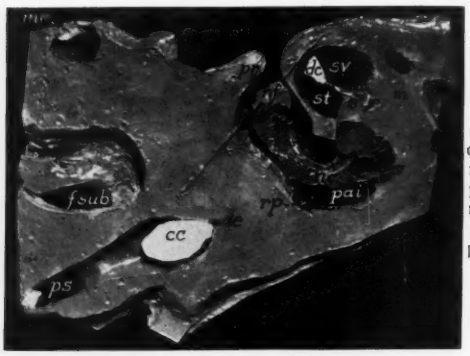

Fig. 6. Block 6, Sec. 235. Nervus facialis is seen progressing toward the tympanum: *cc*, crus commune; *pr*, osselet (Augier); *rp*, ramus posterior vestibularis.

Fig. 7. Block 5, Sec. 264. This block shows the superior level of utriculus and sacculus; *cf* canalis facialis; *cvs*, canal for nervus vestibularis superior; *du*, ductus utriculo-saccularis; *fs*, foramen solitarius; *u*, utriculus.

superior and horizontal canals. The approach of a branch to the posterior canal via the foramen singulare is also shown. On the lateral surface of the pars petrosa (Sec. 264, Fig. 7), posterolateral to the location of the geniculate ganglion, occurs a rounded protuberance of bone which is separated from the main capsule for a good part of its extent. (Microscopic observation shows a quite different staining reaction between it and the main capsule at the point of fusion. This bony protuberance apparently helps to protect the geniculate ganglion which at this age lies exposed within the middle cranial fossa; see Fig. 7, *pr.*) By studying the reverse side of this block we get an excellent conception of the relative bulk of the saccule, utricle and superior ampulla. Upon

Fig. 8. Block 5. Inferior view showing utriculo-saccular duct.

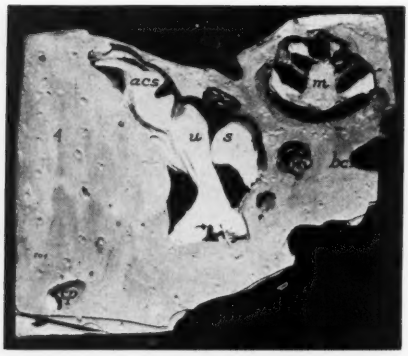

Fig. 9. Block 4, Sec. 304. Sacculus utriculus, and ampulla canalis superioris: *acs*, ampulla canalis superioris; *bc*, basal coil of cochlea; *s*, *sacculus*.

viewing the ampulla in the model, one is reminded of Retzius' description, "biscuit shaped." The termination of the utriculosaccular duct is also indicated *(du)*.

Block 4 (Sections 304-343, Fig. 9). The ampulla of the horizontal canal, the mediolateral extent of the utricle, the saccule, and the maculæ are all clearly presented in this block. The macula of the utricle is here seen, at least in one limb, to lie parallel to that of the saccule. The peripheral parts of the utricle diverge into the canalis lateralis (nonampullated end) and the ampullated end of the canalis posterior, respectively. The location of the macula neglecta is indicated by the arrow. In this specimen the organ is quite rudimentary, appearing merely as a blunt redupli-

Fig. 10. Block 3, Sec. 344. The extent of the cisterna perilymphatica. *cps*, is shown; *acp*, ampulla canalis semicircularis posterioris; *clm*, medial limb, canalis semicircularis lateralis; *ru*, recessus utriculi.

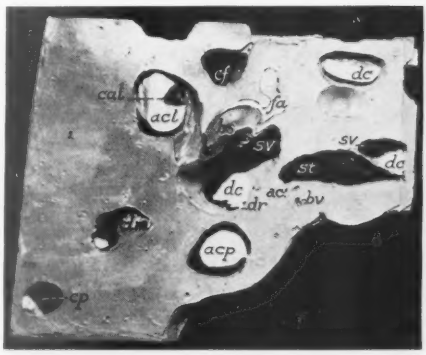

Fig. 11. Block 2, Sec. 375. Showing foot-plate of stapes. Note the irregularity in contour of the nonampullated limb of canalis lateralis. *clm: acl*, ampula canalis lateralis; *ac*, canaliculus cochleæ; *cal*, crista ampullaris lateralis; *bs*, basis stapedius; *dr*, ductus reuniens; *fa*, fossula antefenestram.

cation of the endolymphatic lining at the divergence of the posterior canal and nonampullated end of the horizontal canal. (An accessory crista not shown in the model occurs in this specimen, as described under "Membranous Labyrinth.")

Block 3 (Sections 344-371, Fig. 10). *Per se* this is a rather uninteresting block, but when applied to Block 2 it reveals the large cisterna perilymphatica. It shows the wide separation of basal turn from the other turns of the cochlea and the irregular contour of the bony horizontal canal.

Block 2 (Sections 375-408, Fig. 11). Here we see the origin of the ductus reuniens in the cochlea *(dr)*, the origin of the cochlear canaliculus *(ac)*, its accompanying vein *(bv)*, and the foot-

Fig. 12. Block 1, Sec. 412. The ductus cochlearis is here shown in the basal turn of the cochlea. Fenestra rotundæ is clearly seen: *asc*, anterior stapedial crus; *ffr*, fossula fenestræ rotundæ; *mts*, membrana tympani secundarii; *psc*, posterior stapedial crust.

plate of the stapes *(bs)*, not yet rotated to a vertical plane but still lying in a horizontal plane. The enclosed portion of the facial nerve is seen at this level. The immaturity of the pars petrosa and incomplete rotation is further evidenced by the fact that the middle cochlear turn is cut at more of a tangent than is the apical turn. The basal turn, being so much longer than the upper turns, does not exhibit this feature.

In Block 1 (Sections 412-435, Figs. 12 and 13) the basal turn of the cochlea and the round window are shown. The secondary tympanic membrane lies in general in the same plane as that of the adult. Its concavity toward the scala tympani, as described by

Fig. 13. Block 1. Inferior surface showing head of stapes and groove of facial canal; *cst*, caput stapedius; *fj*, fossa jugularis; *p*, promontorium; *sit*, sinus tympani.

Retzius, is obvious (Fig. 12, *mts)*. In this specimen it actually abuts against the osseous lamina of the basal turn. The facial nerve is seen in its partially closed facial canal. Since everything is in its true relative position, this nerve lies lateral to the stapes and not superior to it as it crosses the middle ear cavity. The sinus tympani is seen on the under surface of the block (Fig. 13, *sit)*. Also in this view we find that the niche of the fenestra rotunda is still in open communication with the fossa jugularis via the fossula petrosa, as indicated by the white arrow in Fig. 13. This communication the author has observed to remain open as late as one year and eight months in a negro infant (No. 4278). The fissure leaves the niche of the round window at approximately the same level as that in which the canaliculus cochleæ leaves the scala tympani. The two fissures run in parallel directions.

The one from the middle ear cavity is not to be confused either with the canal for the vein accompanying the canaliculus cochleæ, or with the cochlear canaliculus itself, as the origin of each of these is within the scala tympani (Fig. 4, *bv*).

The model stacked in toto reveals certain interesting anatomic features. Paramount among these is the depth of the subarcuate fossa, which, although shown in the model as a cavity, is solidly filled with dura. The fossa finally abuts against a huge mass of cartilage, into which the connective tissue of the dura and its accompanying vessels penetrate. Another feature which the model reveals is the extent of the cisterna perilymphatica. This is apparent when Blocks 2, 3 and 4 are studied in relation to each other. The basal turn of the spiral lamina is seen as it descends into the vestibule, and the wide space between the utricular wall and the foot plate of the stapes is obvious.

This model also reveals a rather common anatomic anomaly—that is, an irregularity in the bony contour of the lateral and superior semicircular canals. It failed to reveal, as is shown farther on, another anatomic anomaly, a minute accessory crista in the posterior ampulla.

II. Observations.

A. Cavum Tympani.

The model having been constructed primarily to demonstrate the relationship of perilymphatic and endolymphatic systems of the auris interna, reveals relatively little of the cavum tympani. Only that part relating to the course of the nervus facialis and the stapes is included. In order to understand fully the orientation of the tympanum, its form, boundaries and contents at this stage, the evidence afforded by the sections not included in the model was sought.

Location.—The tympanum of the 6-month, premature infant is inferiorly placed in relation to the otic capsule. In the series studied the tympanum does not appear until Section 374, when the very first tangential cut of the mesenchyme-containing cavity is made. Fig. 11 shows this level of the model and demonstrates the fact that this is the level of the basis stapedis, which here appears with its inner face pointing upward. Figs. 12 and 13

show that the crura are subtended in the tympanum instead of lying in the more horizontal plane familiar in the adult.

The inferior location of the tympanum and the subtended stapedial crura were further verified, for a fetus of this age, by dissection. A postauricular skin incision was made and the subcutaneous tissue dissected so that the auricle and surrounding cutaneous tissue could be reflected forward. On the left side only sufficient further dissection was done to reveal clearly the membrana tympani and annulus tympanicus. On the right side the membrana tympani was removed and also the gelatinous mesenchyme within the tympanum, until the caput and crura stapedis were exposed. By sighting along the basis nasi and the lobulus auriculi and leveling the position of the two auricular lobes, the head can be placed in the Frankfurt plane and fixed for further study. Then by reflecting the dissected auricular flaps, without changing this position, one readily sees on the one side the almost horizontal position of the membrana tympani, on the other the subtended position of the caput and crura of the stapes.

Further relationships which differ from the adult conditions are seen when we observe Fig. 1, Section 304. This is taken at the level of the superior ampulla, through saccule and utricle and almost striking the modiolar axis of the cochlea. A cut made in the same direction in the adult at this level would reveal also the contents of the epitympanic recess, probably a bit of malleo-incudal joint and the upper border of the membrana tympani. Such is not the case at this age. These epitympanic features do not appear in the serial sections until a considerably lower level is reached. In horizontal sections through the region where in the adult we should expect to find these features of the middle ear cavity, there appears in the 6-month fetal ear the cranial cavity with its dural lining.

Shape.—The cavum tympani of the premature infant of this age is cleft-shaped, being quite broad postero-inferiorly. Perhaps "wedge-shaped," with the sharp part of the wedge upward, describes it better. The superior limit of the cavity is irregular, projecting and terminating in a series of three truncated cones. These are filled with mesenchyme. The most lofty of these terminates posterolateral to the nervus facialis and ampulla semicircularis

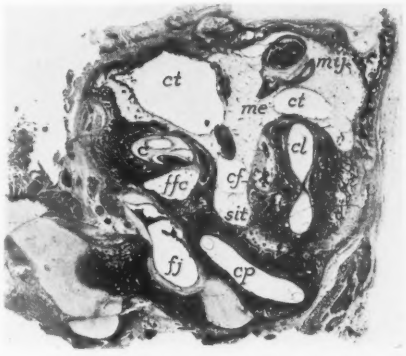

Fig. 14. Section 466. Cavum tympani containing mesenchyme. Rugæ on the extreme left of cavum tympani indicate approach to auditory tube: *mij*, malleoincudal joint. x4-½.

lateralis. This occurs, as previously mentioned in Section 374 (Fig. 11). The two other cones or extensions of the tympanum lie anterior to this one. Both reach the same level superiorly. They lie in the fossulæ fenestræ rotundæ and ovalis, respectively. This makes obvious the fact that the promontory and a part of the vestibular wall of the otic capsule form a large part of the superior boundary of the tympanum at this age.

Walls.—The medial wall (Fig. 14) is highly irregular. It is determined in the anterior part by the cochlea projecting into the middle ear cavity, the form of which is impressed by the contour of its coils. The part of the medial wall separating the basal turn of the cochlea from the tympanic cavity is an extremely thin yet

Fig. 15. Section 497. Cavum tympani with mesenchyme in fossula fenestræ cochleæ. Note stapedius muscle, *ms*; *cos*, caput os stapedius.

double layer of compact bone with an intervening layer of spongy bone containing marrow. From Section 424 on, proceeding inferiorly, the medial wall is incomplete. It is made so by the presence of an elongated channel (the fossula petrosa of Hyrtl) leading to the petrosal ganglion. The fossula petrosa runs in general in the same direction as the cochlear aqueduct.

The posterior wall presents two regions in different planes, a medial and a lateral region (Figs. 14 and 15). The medial region lies on a more anterior plane and is formed by the prominence of the canalis semicircularis lateralis. It is identified as the posterior part of the medial wall of the adult tympanum. The pyramidal eminence consists of dense fibrous tissue and is not yet completely ossified, therefore is not described here as a feature of the posterior wall. The lateral region of the posterior wall is formed by the squamosa, so far as these sections show. The material is not sectioned inferiorly far enough to show the origin of the chorda tympani.

The lateral wall (Figs. 14 and 15) separates the tympanum from the middle cranial fossa. It is of double-layered marrow containing bone and is identified as the tegmen tympani. On its superior level it bears a rounded crest which bulges into the middle ear cavity, projecting slightly anteriorly. This occurs approximately between Sections 390 and 412—that is, it is 0.44 mm. long. It bears no relation to the head of the malleus nor to the body of the incus. The head of the malleus appears in Section 431, where a second similar crest appears and projects toward the head of the malleus. A marked condensation of mesenchyme around the head of the malleus, particularly on the medial side, and extending toward the crest, indicates the future site of the ligamentous tissue. The body of the incus, which appears first in Section 450, shows no connection with tympanic walls.

The lateral wall articulates posteriorly by suture with the squama temporalis. Anteriorly in its superior level, it ends in dense connective tissue without articulation. It lies in close proximity to the musculus tensor tympani and forms a part of the bony support for this muscle and the nervus facialis, as described below. Inferiorly it terminates bluntly in an incompletely ossified portion anterior to which passes the chorda tympani as it leaves

the tympanum. Inferior to the chorda tympani we find dense connective tissue leading to the malleus, and anterior to it a bony process, identified as the processus anterior of the malleus (Section 492). Still further inferiorly the lateral wall becomes completely overlapped by the squamosa. Anteriorly it articulates with the annulus tympanicus.

The inferior boundary is given by the membrana tympani. This does not lie in a perfectly horizontal plane, as is seen by the tangential cut of the sections. It extends from a superolateral position to an anteromedial inferior one. This may be seen in the gross dissection. The annulus tympanicus which supports the membrana tympani and from which the middle or fibrous layer arises, articulates intimately with the otic capsule in certain regions; in others, it is quite widely separated from it. This fact is shown on the sections of the left ear where the block was so oriented as to have the membrana tympani cut in cross-section. The annulus tympanicus is closely adherent antero-inferomedially. Anteriorly it separates from the capsule at the level of the tympanic orifice of the tuba auditiva. Posteriorly it separates from it at the emergence from the capsule of the cartilage of the styloid process and exit of the nervus facialis. No processus mastoidea is developed at this age. At Section 524 a cartilaginous island appears in the posterior part of the floor of the tympanum. Superiorly this is rounded, but at lower levels it becomes ovoid. It remains isolated from the neighboring bony wall so far as the sections continue and lies lateral to the facial nerve. It is interpreted as the styloid process.

Anteriorly the cavum tympani ends superiorly in the orifice of the auditory tube. Below this level no distinct anterior wall exists as yet.

Contents.—Certain measurements were taken in specimen No. 3887 at selected levels for comparison with older specimens. It is recognized that these comparisons can have only relative value, since the possibility of error in the location of identical points in sections from different specimens is considerable. The measurements were taken on the left ear.

The distance from the footplate of the stapes to the tympanic membrane, in a plane at right angles to the footplate, was 3.5

mm. The distance to the head of the stapes was 1 mm. The distance between the round window and the drum membrane in the posterior inferior quadrant, where in older specimens the incision for paracentesis would presumably be made, was 2 mm. The niche of the round window at this stage of development is completely filled with mesenchyme, so that these measurements do not indicate the distance across free space. The actual transverse measurement of pneumatized space is only 1.092 mm. The mucosa over the tip of the cochlea is .5 mm. thick.

For comparison, the distance from drum membrane to stapedial footplate in a 6-month infant was 6 mm.; in a 10½-month infant 4.5 mm., and in an adult 5 mm.

No antrum had as yet developed in specimen No. 3887. Specimen No. 3937, aged 10 days, has a well developed antrum, in which the resorption process has progressed rapidly. The antrum measures 6 mm. transversely at the widest point mounted in the sections. This infant, although born at term, was classed as premature. No hypotympanum had as yet developed.

Ossicles.—The malleus measures 7.5 mm. from the tip of the manubrium to the upper (posterior) edge of the caput as seen in Sections 509-510. Thus the ossicle is at least of this length and may be longer if it could be measured in toto. The manubrium consists of cartilage, but the caput is of marrow-filled bone. The processus anterior (Folii) consists of mature bone (Sections 490-497) and is completely fused with the caput. Meckel's cartilage was not observed, even in rudimentary state, although it was very conspicuous in the 3 months fetus. The processus lateralis mallei was not observed. The sections do not extend inferiorly far enough to permit complete study of this region. On the left side the processus lateralis may be observed as a cartilaginous protuberance which bulges into the external auditory canal carrying the membrana tympani with it. On the medial side of the malleus at this level may be seen the closely associated chorda tympani. A layer of cartilage, 0.157 mm. thick, covers the articular surface of the malleus. A definite joint cavity is seen at the malleo-incudal articulation.

The incus is completely ossified throughout the body and the short and long processes. Like the malleus, it contains a great

deal of marrow around which the ossification forms a shell of compact bony tissue of varying density. The density is greatest in the processes. The incus measures 6.5 mm. from the posterior (upper) edge of the body to the tip of the lenticular process. This latter process appears here as an integral part of the long process and not as a separate bone. Even the marrow cavity is continuous with that of the processus longus. The articular surface for the malleus is cartilaginous as in the malleus and is of approximately the same thickness (0.157 mm.). The cartilage of the lenticular process is located only on the medial (later, inferior) surface. Around this the cartilage of the head of the stapes fits like a cap (Sections 486-492).

The stapes in a specimen of this age assumes quite a different position from that of the adult specimen. As has already been stated, the footplate faces upward. This structure measures 2.8 mm. by 1.28 mm. Its center is of dense bone marrow. Surrounding this is a very thin layer of bone, and around this, in turn, is a layer of cartilage 0.105 mm. in thickness, which fits into the cartilaginous rim of the oval window. Just within the stapedial cartilaginous rim are numerous osteoclasts. The crura, as already pointed out, are subtended. They appear irregularly rounded in cross-section, not yet having acquired the hollow-beam structure of the mature bone. Each crus consists of an incomplete bony lamella which encloses marrow and is surrounded by a very dense layer of fibrous tissue. This is as thick and in some places thicker than the bony lamella. Numerous osteoclasts occur on the outer surface of the bone, while on the inner is a definite alignment of osteoblasts, all lying at right angles to the long axis of the surface of the bone. The caput is still cartilaginous from the level where the musculus stapedius attaches to the place of articulation with the incus.

Muscles.—The stapedius muscle, at this age, is completely surrounded by a fibrous sheath of pyramidal shape, the bony pyramidal eminence not having formed as yet. The fibers of the muscle arise from the posterior, lateral and medial walls of this sheath, run diagonally forward, and are inserted at various levels into a tendon on the anterior aspect of the muscle, which in turn is inserted on the lower part of the head of the stapes. The indi-

vidual fibers of the muscle are as yet not very numerous and are not very closely packed together. Their striations are coarse, the individual sarcomeres measuring 10.5 μ. The muscle as a whole measures approximately 2 mm. in length. (It may here be stated that in a 3 months fetus the fibers of the stapedius muscle had just begun to appear. The type of muscle structure is not yet differentiated.) The stapedial nerve is seen entering the muscle in Sections 520-521. The nerve enters the muscle from the infero-postero-lateral region and is accompanied by a venule and arteriolar capillary. The nerve is first seen separating from the main facial in Section 477. Since the sections are 20 micra thick, that means that the length of the isolated nerve is 0.86 mm.; in thickness it is 0.04 mm.

The tensor tympani appears as a bipinnate structure at the apex of the cochlea in Section 414. It extends in horizontal plane, anteroposteriorly. Here it measures 5 mm. in length. Medial to it is seen the cartilage of the auditory tube, from which it is separated by a double layer of dense fascia. It shows no indication of sending its fibers through this fascia to attach directly on the tube. Like those of the stapedius, the fibrils are striated. Section 384 shows a small twig from the facial entering the semicanal of the tensor tympani. It cannot be seen actually entering the muscle fibers. No branch from the trigeminal nerve nor from the otic ganglion was identified. No muscle fibers were observed crossing from this muscle into the m. tensor veli palatini.

It may be noted here that the musculus tensor tympani was observed in embryo X, estimated to be less than 2 months. In a 3 months fetus it is a well-developed muscle (specimen XXI).

Pyramidal Eminence.—The pyramid lies on the anterior of the two regions of the posterior wall, just anterior to the horizontal semicircular canal. As indicated above, the pyramidal shape is at this age maintained by dense fibrous tissue. This shows signs of ossification in just two regions. These are not isolated centers of ossification but are extensions from the bony wall of the lateral semicircular canal. As yet they may be said to protect the facial nerve more than they do the muscle. One ossified region lies posterior to the tendon of the muscle as it leaves the connective tissue sheath. The other lies supero-anterior to

the body of the muscle, forming a thin shelf projecting toward the tympanum. Inferiorly there is no bony protection. The cartilage of what is interpreted to be the stylomastoid process appears between the base of the muscle and the tympanum. The fibrous sheath surrounding the muscle separates the body of the muscle from the facial nerve. On the anteromedial side this sheath arises in part from the pit of the sinus tympani.

Sinus Tympani.—The sinus tympani (Fig. 14) is a deep ovoid depression extending into the depths of the otic capsule between the lateral and posterior semicircular canals and anteromedial to the pyramid. At this age the sinus is filled with mesenchyme. In fact, as is shown later, it is one of the pockets in which mesenchyme remains longest. The extent of the sinus can be traced by a circuitous route through the capsule posteriorly until it becomes continuous with the potential parantral regions. Complete ossification will later block this path.

Nervus Facialis.—The nervus facialis appears to enter the tympanum in a closed canal, as seen in Fig. 11, Section 375. This, however, is merely the course of the nerve through the bony floor of the skull. It is the only place where the nerve is completely surrounded by bone at this age. This bone is 0.76 mm. thick. It maintains toward the dural side a slight aperture for the passage of a nerve branch and blood vessels. The canal definitely opens toward the tympanum in Section 412 (Fig. 12). It is here seen that the nerve does not enter the tympanum until much farther on its course than is the case in older specimens. As it courses posteriorly across the tympanum it lies in an open groove upon a bony support. Posteriorly the bony support is continuous with the very prominent bony contour of the canalis semicircularis lateralis (Fig. 12, *cf*). Anteriorly this bone forms the semicanal for the tensor tympani. The canals for these two organs are not wholly separated from each other. This bony support did not occur on the left side. It is interpreted to be a persistent parotic crest and not a suprafacial commissure (Figs. 13 and 14). It is to be recalled that the model, being a negative, reverses the orientation for the sections. As the facial nerve takes its descending course it is partly exposed. More inferiorly it is protected on its anterior surface solely by the presence of the stapedius muscle

for much of its course. At Section 483 the incomplete wall of the pyramid partially encloses the nerve. At Section 497 (Fig. 15) the bony wall of the pyramid disappears, grading into dense connective tissue which encloses the stapedius muscle. From this point until Section 524 the nerve is protected anteriorly by this muscle. Then a cartilaginous island, ovoid in shape, appears anterolaterally. This is interpreted as the styloid process. Nerve and cartilage proceed together in their downward course, the cartilage coming to lie more and more laterally. Finally at that point where the drum membrane with its annulus tympanicus is but loosely applied to the otic capsule, the two structures, cartilage and nerve, emerge from the tympanum. Gross dissection of specimens readily shows how superficial and close to the edge of the drum membrane lies the stylomastoid foramen. The sections show the stylomastoid artery with its accompanying vein surrounding the nerve and giving the nerve abundant blood supply. Direct connections between the vein and the neighboring bone marrow were traced.

The branches of the facial nerve will now be described. As noted above, the stapedial nerve separates from the facial in Section 477. It is completely bound in its own sheath and runs for 0.86 mm., whereupon it enters the muscle accompanied by a venule and an arteriolar capillary (Sections 520 and 521). The origin of the chorda tympani is not seen in the right ear of this series as the block is incomplete. The nerve is seen, however, coursing in mesenchyme between the malleus and incus. It follows the processus anterior and in Section 484 passes out of the tympanum through the iter chorda anterius (Glaserian fissure) inferior (lateral) to the processus anterior. The course of the facial nerve will be described in detail in the section on the pars petrosa.

Auditory Tube.—The tuba auditiva was not modeled, as only part of it was included in the specimen of the right side. It is well seen almost in toto on the left, however, and on both right and left the cartilage extends toward the tympanum to the apex of the cochlea. No osseous tube exists at this age. The lumen exhibits clefts and rugæ. It is lined with tall ciliated columnar cells between which are intercalated goblet cells. On the left side a slight constriction of the lumen may be seen, somewhat below

the level of the cochlea. Scattered mucus glands occur near the tympanic orifice.

A study of the younger specimens was made in order to observe the development of the tube. Specimen X, estimated to be less than two months, shows a slitlike auditory tube patent at the level of the apex of the cochlea. No well-defined epithelial lining of the tube was observed, although evidences of an alignment of large cubical cells could be seen.

In specimen XV M256, estimated to be 2¾ months embryonic life, epithelium is formed in the auditory tube but not in the middle ear cavity. The respiratory parts of the nasal cavities in this specimen are well lined with high ciliated columnar epithelium. The auditory tube shows a tendency towards a constriction or narrowing near the middle of its course. The lumen at the narrowed point measures 0.02 mm. in vertical plane and 0.062 mm. in the plane of the section. The tube is 2 mm. long.

An embryo, 3 months old, cut to include the whole head in cross-section, clearly shows the fibers of the tensor veli palatini and of the levator palatini. These muscles are well developed, although their association with the tube is as yet not very intimate. No striations were observed in these muscles at this age. No cartilage had appeared along the tube.

A 4 months fetus (XVII M277), sectioned in the vertical plane, presents some features of interest concerning the auditory tube. The cut was directed at right angles to the long axis of the pars petrosa. The medial cartilage of the tube appears 0.2 mm. above the pharynx and exhibits four centers of chondrification. There is some condensation of mesenchyme in the region of the superior cartilage. The auditory tube shows a definite torsion in its course. At the pharyngeal end the long axis of the lumen lies in the horizontal plane. Its short axis, measuring 0.25 mm., is in the vertical plane. As the tube is followed toward the tympanum a shift in cross-sectional axes can be observed. At the narrowest point in the tube the vertical axis measures 0.65 mm., the horizontal 0.39 mm. Cartilage accompanies the tube for 2 mm. of its course; it does not extend so far as the level of the cochlear apex, and no bone appears in this tubal region. Epithelium lining the lumen shows differentiation in different sides of the tube. Peculiarly

enough, there is more indication of rugæ midway in the course of the tube than at either end. At the pharyngeal end numerous glands of mixed type (containing demilunes) are observed.

A specimen estimated to be 4½ months fetal life shows ciliated epithelium on the roof as well as on the floor of the auditory tube. Towards the tympanic end the epithelium flattens out into cuboidal and then into cells that are practically squamous, over the apex of the cochlea. The ciliated cells where present are not so well differentiated as are those of the pharynx. Goblet cells are present. No indication of adenoid tissue was observed in any of these young specimens.

Mesenchyme.—The cavum tympani of the 6 months premature infant is well filled with mesenchyme (Fig. 14, *me*). The parts of the ossicles contained in the epitympanum are almost surrounded by this embryonic tissue. The same tissue fills the fossula fenestræ cochleæ, the sinus tympani, and the space between the stapedial crura. Histologically, the mesenchyme presents a structure typical for a fetus of this age. It consists of large cells whose nuclei are granular and stain with hematoxylin. In some cells the nuclei are long, narrow and spindle shaped; in others, they are more rounded. In section these cells appear, for the most part, triangular in shape. Their form is probably pyramidal. Their cytoplasm is pink-staining (H and E) and extends from the main body of the cell in long processes which comprise the meshwork of the mesenchyme. Between the processes is a pink-staining ground substance.

Scattered through this tissue are numerous large cells with granular cytoplasm, which are decidedly irregular in shape, thus suggesting that these cells have had amoeboid movement. Numerous capillaries are found throughout the mesenchyme. A single layer of low cuboidal epithelial cells lines the pneumatized areas. Toward the region of the auditory tube the epithelium becomes columnar and finally ciliated columnar. Certain small cavities in the mesenchyme (Figs. 14 and 15) which eventually coalesce in the process of air space formation are not yet lined by epithelium.

In the region of the auditory tube there is no trace of mesenchyme, nor is there any along the drum membrane. Air spaces

have formed lateral to the body of the incus (lateral epitympanic recess), medial to the manubrium of the malleus (Figs. 14 and 15), and also around the anterior crus of the stapes. There is evidence of a slightly greater degree of pneumatization of this delicate tissue in the right ear than in the left.

A comparative study of mesenchymal remnants in prenatal, infant and adult specimens from forty-two cases was made. The following table presents the data.

This table shows that no pockets of mesenchyme were observed in any specimen older than thirteen months. A thickened mucoperiosteum may persist longer. Places where mesenchyme remains longest are in the hypotympanum, the par-antral cells, the fossulæ (niches) of the oval and round windows, in the epitympanum, and in the sinus tympani. The age of specimen No. 4151, it will be noted, is 8 fetal months. This child lived only one day. For its degree of prematurity there is relatively little mesenchyme. On the other hand, specimen No. 4232, a baby aged 2½ months, shows large masses of mesenchyme in the epitympanum. However, this infant weighed but 2051 gm. at birth and therefore was probably undeveloped at birth.

Membrana Tympani.—The membrana tympani of specimen No. 3887 is best studied from material from the left ear. Here the block was so cut as to present the membrane in cross-section. At this age the three component layers are readily distinguished. The ectodermal layer near the periphery consists of five rows of flattened rectangular cells. The number of rows decreases toward the center of the membrane to three or even two layers. No stratum granulosum can be distinguished on the medial side of the epidermis. No pigment is observed. Both of these features are readily observed in the epidermis of the neighboring external auditory canal where pigment is quite abundant.

The middle or fibrous layer quite obviously takes its origin from the annulus tympanicus. A heavy layer of connective tissue arises from the periosteum of the outer perimeter of the ring. This structure appears horseshoe shaped in section. Just central to the tips of the horseshoe, the layer from around one arm joins the layer from around the other arm. It is as if the annulus lay like a ring, whose inner surface is hollowed, in a double fold

TABLE I. INCIDENCE OF MESENCHYMAL REMNANTS*.

No.	Age	Race	Sex	F. F. C.	F. F. V.	E.	H.	S. I. T.	A.	Pa.	Fibrillar Con. Tissue
XVI	7½ fetal months	W	F	+	+	+	+	+	+	not formed	
4151	26½ hours 8 fetal mos.	W	F	+	+	+	+	0	0	m	
3924	Stillborn 4 da. prem.	N	M	+	+	+	+	+	0	m	
4331	1 day	N	M	very slight	0	+	+		0	m	
3937	10 days	W	F	slight	slight	+	+	0	0	+	
4439	15 days	W	M	+	+	+	+	+		not seen	
4306	21 days	W	M	slight	slight	not seen	+	0	not seen	not seen	+
3849	1 month	N	M	slight	very slight	not seen	+	+	0	m	
4330	1 month 27 days	W	M	+	+	+	+	+	0	m	+
3967	2 months	W	M	+	+	+	+	+	0	m	+
4427	2½ months	W	F	+	+		+	+	0	+	
4232	2½ months	N	M	+	+	+	+	+	0	+	+
3949	2½ months	W	F	0	0	not seen	+	0	0	0	
3873	3½ months	W	F	0	+	not seen	not seen	+	+	+	slight

3865	4 months	W	M	+	+	+	not seen	+	+	+	F. F. C. and F. F. V.
4078	5 months	W	M	+	+	tmp	+	0	+	+	+
3920	5½ months	W	F	left side +	0	0	0	+	0	+	
3947	7 months	N	F	+	0	tmp	not seen	+	0	+	+
3923	10½ months	N	M	0	0	0	0	not seen	0	0	
4106	1 year	N	F	0	0	0	0	0	0	0	+
4338	1 year	W	F		+	+	tmp	+	0	+	+
4048	13 months	W	M	+	+	tmp	tmp	fat	0	tmp	+
II	13 months	W	F	0	0	0	0	0	0	0	+
3878	1 year 4 months	W	F	0	0	0	0	tmp	0	tmp	
4278	1 year 8 months	N	F	0	0	0	0	s. t.	0	tmp	+
4457	2 years	W	F	0	0	0	0	0	0	0	
3951	5 years	W	F	0	0	0	0	not seen	0	0	Petrous T. T. + A lft
3903	8 years	W	M	0	0	0	0	fat	0	0	F. F. C. and F. F. V.
3956	10 years	W	F	0	0	0	0	0	0	0	
4336	12 years	W	M	0	0	0	0	0	0	0	
3948	13 years	N	M	0	0	0	0	0	0	0	

TABLE 1. INCIDENCE OF MESENCHYMAL REMNANTS.*—Continued.

No	Age	Race	Sex	F. F. C.	F. F. V.	E.	H.	S. I. T.	A.	Pa.	Fibrillar Con. Tissue
4056	31 years	N	M	0	0	0	0	0	0	0	F. F. C. and F. F. V.
3884	35 years	W	M	0	0	0	0	0	0	0	
4486	40 years	N	F	0	0	0	0	0	0	0	
4169	42 years	W	M	0	0	0	0	0	0	0	
3972	44 years	W	M	0	0	0	0	0	0	0	
3897	45 years	W	F	0	0	0	0	0	0	0	F. F. C.
3917	47 years	N	F	0	0	0	0	0	0	0	
4098	48 years	N	F	0	0	0	0	0	0	0	F. F. C.
3894	51 years	W	M	0	0	0	0	0	0	0	F. F. C.
4152	69 years	N	F	0	0	0	0	0	0	0	
4164	73 years	W	M	0	0	0	0	0	0	0	

Marrow occupies the areas of the future parantral cells until the middle of the third month. Thickened mucoperiosteum as contrasted with actual masses of mesenchyme may persist until the middle of the second year, or in pathologic cases may form in the adult.

*The letters F. F. C. refer to fossula fenestrae cochleae; F. F. V., fossula fenestrae vestibuli; E., epitympanum; H., hypotympanum; S. I. T., sinus tympani; A., antrum; Pa., parantral cells; tmp., thickened mucoperiosteum; m., marrow.

of membrane. The membrane arises not only from the periphery of the ring, looping around it as it were, but also arises from the perimeter of the hollow of the ring. The fibers arising here appear much more delicate than those in the outer layer. They immediately take a concentric course and are enclosed within the double outer layer. At this age all these fibers are radiating. No circular fibers could be distinguished as yet. Very minute nuclei, observed really within the component cells of the mucosal layer, may possibly represent cut nuclei of the developing circular layer.

The mucosal layer of the drum membrane presents in cross-section three rows of cells. Near the periphery of the drum these cells become decidedly cuboidal. They continue into a deep sulcus in the middle ear adjacent to the tympanic ring and then over the surface of that cavity. Upon approaching the center of the drum membrane these cells become more and more flattened until finally they are so definitely squamous that the nuclei bulge prominently into the tympanum, while the protoplasm tapers off to meet that of the contiguous cells. As noted above, very minute nuclei are occasionally interspersed among these cells.

Barring the obviously thickened periphery, the drum membrane varies in thickness in different regions. Opposite the footplate of the stapes it measures 0.05 mm.; opposite the promontory in the same section it measures 0.1 mm. The cross-section of a capillary lying between the epidermis and the fibrous layer measures 0.025 mm. At this level the blood vessel supplying the anterior part of the drum membrane lies between the epidermis and the fibrous layer, while the vessel in the posterior half is seen lying between the mucosa and the fibrous layer. The mucosa is relatively less well developed.

The annulus tympanicus presents an excellent example of the contrast in structure and activity of osteoclasts and osteoblasts. This bone is ossified at an extremely early period in embryonic life, beginning late in the second month. The process of growth therefore necessitates the removal and reshaping of bone already deposited. This is being done in specimen No. 3887 by the osteoclasts which lie along the concavity of the annulus. As many as eleven osteoclasts were counted lying within the semicircular cross-section of the annulus in one section. These cells have the

typical pink-staining (eosin) cytoplasm and contain as many as five to fifteen nuclei. On the outside border of the tympanic ring are crowded the numerous osteoblasts which build up the new bone. They are blue-staining (H. and E.) throughout and show relatively little cytoplasm.

Some observations on the membrana tympani in younger specimens were made. This organ has already begun to appear in the embryo of less than two months. Condensation and alignment of the mesenchymal cells mark its course. The annulus tympanicus is already composed of bone, although the otic capsule is hyaline cartilage as yet. The annulus has not yet developed the grooved internal contour so that it does not appear horseshoe shaped in cross-section.

In the 3 months fetus in which the whole head was sectioned in horizontal plane it is interesting to note that the superior part of the tympanic membrane is directed laterally. It first comes into view at the level of the oval and round windows of the capsule, indicating a relatively inferior position. Epidermis is already well differentiated. Annulus tympanicus is not yet horseshoe shaped in section.

Specimen XVII (less than 4 months fetal life) shows the three types of tissue in the membrana tympani well differentiated. The annulus here is hollowed out as in the older specimens.

(The second, concluding, installment will appear in the March issue.)

XCI.

The Cause of Otosclerosis.*

ONTOGENESIS OF THE AURAL CAPSULE.

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In the previous chapter, the development of the labyrinthine capsule was followed from the condensation of mesenchyme about the otic vesicle (fourth week) to the period of beginning ossification (fifteenth to sixteenth week). Differentiation of mesenchyme into precartilage, dedifferentiation of precartilage into syncytial reticulum around the membranous labyrinth, differentiation of precartilage into cartilage, disappearance of syncytial reticulum to form the periotic spaces (cisterna, scalæ, etc.) were all considered.

OSSIFICATION OF THE CAPSULE.

Next we are to study the growth of the otic capsule from the beginning of ossification to its completion (in addition, further facts from mesenchyme to cartilage will be discussed). In the aural capsule, at or just before the sixteenth week, ossification begins. Just as in the case of flat membrane bones and long cartilage bones, the appearance of bone in the capsule of the ear is in certain definite centers which later fuse.

According to Bast:[1] The first center of ossification appears in the outer part of the capsule on the beginning of the first turn of the cochlea just anteroventral and medial to the round window. Its greatest growth follows the growth direction of the cochlear

*From the Oscar Johnson Institute of Washington University.

This section should be read with the illustrations appearing in "The Development of the Organ of Hearing." ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Vol. XL, No. 3, Sept., 1931, p. 812.

duct. This first center unites with another center on the upper aspect of the cochlea. In a 16 3/7 week human fetus this center is well advanced. Center No. 2 lies on the outer part of the capsule on the inferomedial side of the ampulla of the posterior canal immediately below the entrance of the nerve to this ampulla. Its appearance is about the same time or a little later than that of No. 1. Center No. 3 begins in the outer part of the capsule and overlies the entrance of the nerves into the ampullæ of the superior and lateral canals; time, same as No. 2 or a little later. Center No. 4 lies superolateral to the round window. Its existence as a constant center is doubtful, since No. 1 and No. 2 could easily fuse without this intervening center because of their close proximity when No. 4 appears. In an 18 weeks human fetus the next eight centers are present. Some of these centers appear at approximately the same time. The fifth center arises in the outer part of the capsule and is located in the lateral wall of the internal auditory meatus. The sixth center arises in the inner part of the capsule overlying the medial branches of the cochlear nerve where they enter the cochlea. Center No. 7 arises in the outer part of the capsule and lies in the upper medial wall of the internal meatus. Center No. 8 begins in the outer part of that portion of the capsule which covers the upper slightly medial aspect of the cochlea overlying the course of the cochlear duct. Center No. 9 arises in the outer part of the capsule at the inferomedial margin of the internal meatus. Center No. 10 arises in the outer part of the capsule overlying the most posterior bend of the superior semicircular canal, a little above the common crus of the superior and posterior canals. Center No. 11 is located in the capsule overlying the superolateral aspect of the cochlea and No. 12, between the cochlea and the upper branch of the vestibular nerve. Just below center 12 there is a bony enlargement, No. 13, which extends downward as a slender arm, partly encircling the vestibular nerve, and then becomes continuous with center No. 2. The last distinctly separate center, No. 14, arises in the outer part of the capsule covering the most posterolateral crus of the posterior canal. The area around the stapedial footplate is one of the last to ossify, particularly the part anterior to the stapes (region of the fissula ante fenestram).

THE CARTILAGE MODEL AND ITS PERICHONDRIUM.

The cartilage model of the aural capsule is surrounded by perichondrium differentiated from mesenchyme. The perichondrium comprises two layers: an outer fibrous one and an inner cellular one of chondroblasts. As we have previously learned, these chondroblasts enlarge, develop protoplasmic processes, the smaller ones disappearing, the larger ones joining to enclose the cartilage cells in lacunæ. Prior to the second embryonic month a red staining substance is enclosed within the lacunæ. This is not the final endoplasm of the cartilage cell but some substance of unknown composition. The cartilage cells[2] contain fat and glycogen. Thread-like mitochondria (sometimes in the form of granules or short rods) are noted. To one side of the nucleus is the Golgi apparatus (coarse network). There are usually two nucleoli present.

Probably the intercellular basophilic substance, known as matrix, is secreted by the cells. At first showing fibrills, the matrix finally becomes homogeneous. It is composed of collagen, chondromucoid, chondro-albumoid and chondroitin-sulphuric acid. Chondromucoid gives rise to much more chondroitin-sulphuric acid upon decomposition than do the mucoids isolated from other types of connective tissue. The basophilic staining of matrix is said to result from the presence of chondroitin-sulphuric acid. Chondromucoid could account for the blue staining. The collagen is acidophilic but its staining is masked by the more prominent basophilic reaction. Even after the fibrils can no longer be noted with the ordinary stains they may be detected by silver impregnation, polarized light, etc. Immediately around the cell is the capsule which is more refractile than the remaining matrix and is supposedly the most recently formed matrix.

Growth of cartilage is by mitotic division of cells in the chondrogenic portion of the perichondrium and by mitotic or amitotic division of cells in the lacunæ. Cartilage regeneration may occur from the perichondrium, from cartilage cells or by metaplasia of adjacent connective tissue cells.

Cartilage shows no blood vessels, nutrition being gained by the passing of tissue fluid from the perichondrium through the matrix. No lymphatics are found in adult cartilage, but interlacunar canaliculi have been noted. There are no nerves in cartilage.

When cartilage cells enlarge (process of ossification) they produce the enzyme phosphatase which probably has to do with calcification. The physical nature of cartilage matrix is such that it invites the deposit of calcium salts from the solutions in which they are found in the body.

DEVELOPMENT OF A CENTER OF OSSIFICATION.

The first change in a center of ossification (about the fourth fetal month) is an enlargement of the cartilage lacunæ and a shrinking of the cartilage cells. Vacuole formation and disappearance of mitochondria may be noted. At this stage some calcium has already been deposited, as cutting the cartilage gives a gritty feeling. Soon ruptures appear in the perichondrium. New thin-walled vessels are formed in the perichondrium (single layer of endothelium). These vessels, accompanied by osteogenic cells, histiocytes, etc., enter the cartilage model through the perichondrial ruptures (Fig. 8). At this stage the osteogenic cells of the inner perichondrial layer are laying down perichondrial bone. This is a fiber bone formation similar in every respect to membrane bone formation elsewhere. A broad band of calcified collagen with nests of osteocytes appears under the perichondrium (periosteum). Later interlacing fiber bone trabeculæ are formed in many layers with primary marrow between the bars. These trabeculæ stain a bright red and show no enclosed cartilage remnants as does the enchondral bone later. While the perichondrial bone is forming the cartilage model is being invaded and rapidly destroyed. Not all the cartilage cells are destroyed. Those that escape are invaded by osteogenic cells (Fig. 10) and calcification ensues. In place of the destroyed cartilage we now find myriads of primary marrow cells. Around the calcifying cartilage lacunæ the matrix is undergoing a metamorphosis into a pink-purple staining osteoid (Fig. 12). With this tissue as an anchor, the osteogenic cells line up and begin to secrete an intercellular red staining matrix. These cells in epithelioid alignment may now be termed osteoblasts and when encased as bone cells are termed osteocytes. Layer after layer is built up around the calcified cartilage rests and their accompanying osteoid. The primary marrow spaces diminish in size to finally become the adult marrow spaces

and Haversian canals. Meanwhile the areas abutting upon the periotic spaces are being transformed into the fiber bone endosteal layer, which, like the perichondrial layer of bone, is free of cartilage rests. The endosteal layer is membrane bone formed from syncytial reticulum cells. The entire enchondral area is extremely vascular. Much of the perichondrial bone is later encroached upon by the Haversian systems formed in the enchondral division. Also many of the original Haversian systems probably break down and are replaced by new ones. Between the Haversian systems, in the adult, is found a fine fibered lamellarless bone.

Osteocytes show protoplasmic processes extending into the canaliculi which pass from the Haversian lamellæ into the Haversian canals and also from periosteal and endosteal bone into enchondral bone. During matrix formation mitochondria are numerous; later they diminish in number. Bone matrix consists of organic and inorganic constituents. The organic, according to Ham,[2] is osein (similar to collagen of other connective tissues), mucoid and an albuminoid (osseomucoid and osseoalbumoid). The inorganic part is 60 to 70 per cent of the total and consists chiefly of calcium and phosphorus with a small amount of magnesium. Just as with cartilage matrix, that of bone is of fibrillar structure cemented by an amorphous substance. The fibrils become arranged in osteocollagenous bundles or lamellæ. The lacunæ are surrounded by a thin structure similar to the capsule of the cartilage cell. It is highly refractile and homogeneous.

Lamellæ are formed by layers of osteoblasts under the periosteum, around the Haversian canals, and in the marrow spaces. The osteoblast takes a basophilic stain, the osteoclast an oxyphilic one. Both the osteoblast and osteoclast differentiate from the osteogenic cell formed in the inner layer of the perichondrium. With phosphotungstic acid a granular appearance of the osteoclast is brought out which is absent in the osteoblast. The cytoplasmic border of the osteoclast tends to be irregular, often showing a comb-like edge with the prongs sticking into the bone which is being resorbed (Howship's lacunæ). The nuclei of osteoclasts often appear pyknotic. Although the osteoclast is believed by some to be purely a phagocyte it does not take the vital stains.

There is a theory that dead bone matrix (for example, prepared bone of otosclerosis) is dissolved by carbon dioxide produced by living cells. Disintegration of bone may cause a foreign body type of reaction, which may explain the formation of osteoclasts (multinuclear giant cells). Ham believes the osteoclast probably aids in bone resorption by producing carbon dioxide and to a limited extent carries on phagocytosis. Halisteresis means the loss by bone of its calcium salts. By creeping replacement of bone and cartilage is meant an invasion by osteoblasts, the formation of new bone and gradual resorption of dead bone or cartilage. In addition to the creeping of osteogenic cells there is in the resorption process also a chemical effect which may be from osteogenic cell secretion or from the tissue fluids. Pressure may also be a factor.

The Haversian canals contain blood vessels, lymphatics, osteoblasts and other cells. The fibrils of the Haversian lamellæ run in different directions to give architectural strength to the bone. Osteocytes are situated in and between the lamellæ. From the bone cells pass small canals into the matrix. They run in all directions and anastamose with those of other cells. Through these canaliculi fluid passes from periost to endost. Collagenous bundles (Sharpey's fibers) pass from periost into the bone matrix and anchor the periosteum to the bone. They are not present in the Haversian systems. The blood supply for the bone is obtained through Volkmann's canals, which pass from the periost at right angles into the compact bone. They connect with the Haversian canals and anastamose with vessels supplying the marrow. Lymphatics are present in the periosteum and accompany the vessels in Volkmann's canals, marrow spaces and Haversian canals.

CALCIFICATION OF MATRIX.

One of the most interesting chapters in osteology is that of calcification of matrix. Calcium taken into the body with food is absorbed from the alimentary tract and distributed by means of the blood stream. The normal calcium content of the blood is greater than could be retained in simple solution. The explanation lies in the effect of parathyroid hormone, which serves to

retain calcium phosphate in solution. Vitamin D, through the agency of parathyroid hormone, increases the blood calcium level. Between the calcium of blood and that of the bones there is normally an equilibrium. In the blood is found an inorganic and an organic calcium compound. Between the two is also, normally, an equilibrium. An excessive supply of parathyroid secretion, as, for example, in adenoma of the parathyroid, leads to the removal of abnormal amounts of calcium salts from the osseous system, thereby raising the blood calcium level and increasing excretion of calcium. This condition is seen in Paget's disease. If the condition is not too advanced the removal of the parathyroid tumor stops the oversupply of parathormone and leads to a recalcification of bone and recovery.

The deposit of calcium in matrix is due to the physicochemical properties of the matrix plus the presence of phosphatase secreted by the bone and cartilage cells. This enzyme is not present in cartilage until a center of ossification appears.

When too little calcium is present in the blood (hypocalcemia) we see in centers of ossification a great profusion of cells and imperfectly calcified matrix or osteoid. Such a condition exists in rachitis. In hypercalcemia there may occur in various parts of the body metastatic calcifications, especially in areas of acid excretion, such as the kidney, lung and gastric mucosa.

FISSULA ANTE FENESTRAM.

The last portion of the aural capsule to ossify is the oval window region, particularly the area anterior to the footplate. This region, known as Cozzolino's zone, is the site of entrance into the tympanum of a passageway from the vestibule, the fissula ante fenestram. In the chapter on phylogenesis the possible origin of this vestigial structure was discussed. It is particularly interesting because in its neighborhood is the site of predilection in otosclerosis. The fissula is present in all human embryos and in most adult temporal bones, according to Bast. It is filled with a loose connective tissue and various types of cells, including undifferentiated mesenchymal elements. It is lined with cartilage, which may persist throughout life.

In a presentation before the American Otological Society, May 8, 1933, the writer stated that possibly the fissula ante fenestram is a homologue of the accessory endolymph duct of the acanthius vulgaris (spine shark) and the raja clavata (thorn ray); also of the ductus fenestræ ovalis of the rana esculenta (edible frog). In his discussion, Dr. J. Gordon Wilson expressed doubt concerning this, because of the fact that in mammalians and other intervening phyla the fissula ante fenestram "is not found." In a personal communication from Dr. Frank B. Hanson, department of biology, Washington University, this authority stated that, despite the fact that the fissula ante fenestram is absent in most mammalians and the other intervening phyla, it is still possible that the structure in man is a homologue of the above mentioned structures in the shark, ray and frog because of the theory held today that man probably did not descend through the type of reptile, etc., in existence today, but rather from a much lower form of reptile-amphibian. The fissula ante fenestram, therefore, may have been transmitted from fish to amphibian, to amphibian-reptile, and so on to man, while most of the reptile to mammalian phyla in existence today have remained without the structure. Dr. T. H. Bast, in a personal communication, expresses doubt concerning the writer's theory of the origin of the fissula ante fenestram, but adds that it is not impossible that the theory is correct.

We read in Bast's monograph "Ossification of the Otic Capsule in Human Fetuses"* the following (page 66): "In his (Hyrtl) Corrosion-Anatomie (1873, page 230), in describing the ear of the 'hyaena spelaea' he writes: 'Before the oval window one finds in the outer wall of the vestibulum a small evagination which in the corrosion appears as a low stump-like process." He says that it is absent in most mammals, but he found it in a young antelope where a diverticulum from the sacculus projected into it. It is of interest that Ibsen showed a similar diverticulum of the utriculus projecting into the evagination posterior to the stapes. Ibsen, on pages 38 and 39, and Fig. 31g of his Anatomiske Undersogelser over Oerets Labyrinths, says that there is a funnel-

*Bast, T. H. (Loc. cit.)

shaped evagination from the vestibule back of the stapes. In Fig. 6 he shows at *g* a diverticulum projecting into this evagination and calls it canalis membranaceus caecus. He found it in twenty-three out of sixty newborn children. In a few cases he could insert a human hair into the opening from the middle ear into the vestibule. He concluded that the structure was a remnant of an embryonic structure, especially since he seldom found it in the adult ears. In 1890, in his Korrosions-Anatomie, page 29, F. Siebenmann writes: "Between the posterior ampulla and posterior end of the oval window there is, in about half of my corrosions—in the newborn much oftener than in the adult—a small pyramid-like structure which projects into the bony capsule." He calls it "fossula post-fenestram." He compares it with the funnel-like structure containing the diverticulum of the utriculus (canalis membranaceus caecus) described by Ibsen. He picures this structure in his figure 19, f. p. f. Again he writes: "Another opening, which is more slit-like, occurs immediately in front of the oval window: fissula ante fenestram, where the vestibule unites with the upper wall of the scala vestibuli." This is pictured in his figures 14 and 20, fi. a. f.

Siebenmann compares this structure with the evagination containing the diverticulum of the sacculus which Hyrtl described, and considers it in man as a rudiment of the evagination found in the hyena and antelope.

We may conclude from the above that J. Gordon Wilson was probably mistaken in his statement that the fissula ante fenestram is found in man alone. Of particular interest is the saccular diverticulum which Hyrtl found in a young antelope, projecting into the fissula. Immediately we recognize the possible relationship between this saccular projection and the accessory endolymph projections found in the shark and the ray. Then, we have Ibsen's report that in twenty-three out of sixty newborn children he found a diverticulum projecting into the fossula post fenestram. He found in a few of these cases that a hair could be passed from tympanum to vestibule through the fossula. Siebenmann compared the fossula postfenestram in his specimens to Ibsen's canalis membranaceus caecus and the fissula ante fenestram to Hyrtl's evagination containing the diverticulum of the sacculus.

He then adds that he considers the structure in man as a rudiment of the evagination found in the hyena and antelope.

Summarizing our data: In the spine shark is a utricular evagination (accessory endolymph channel) passing as far as the outer capsule wall. In the thorn ray a similar utricular diverticulum passes to and through the outer capsule wall. In the frog a passageway, no longer connected with the utriculus, passes from the perilymph chambers, through the outer capsule wall between fovea fenestræ ovalis and footplate of columella, to end as a blind sac in the tympanum. In the hyena and antelope there is an evagination in the outer wall of the vestibulum. In the antelope a diverticulum of the sacculus protrudes into the evagination. In children, Ibsen found a capsular evagination posterior to the stapes in one out of three new-born children. In this evagination was a diverticulum. Siebenmann finding the evaginations concluded that the fissula ante fenestram in man is a rudiment of the evagination found in the hyena and antelope.

Although nothing is proven, the above data certainly present the basis for interesting speculation, and since otosclerosis has as its site of predilection the region of the fissula ante fenestram, seemingly a vestigial structure, the theory of regression in otosclerosis would seem to be strengthened. Tracing otosclerosis backward is a difficult task but an intriguing one. It was not many years ago that otosclerosis "started" at puberty or later. Then came Siebenmann's and O. Mayer's cases,[3] each 5 years of age; in 1915, Manasse's case, 3½ years of age; in 1930, Guild's case, 1 year and 10 days of age. In June of 1933, the writer came upon the histologic picture of otosclerosis in a 7 months human fetus, the first case of otosclerosis ever found before birth. This case will be reported fully in the chapter on pathogenesis of otosclerosis in the present work. In the department of embryology of the Oscar Johnson Institute of Washington University, a 2 months human embryo of an otosclerotic mother has just been serially sectioned. It is hoped that in this precartilage stage something may be found which will throw further light upon our problem. The mother was observed for a long period of time by Dr. French Hansel, who reports classical otosclerosis. She

was then studied audiometrically by Dr. Cordia Bunch, who reports findings typical of otosclerosis.

GENERALIZED BONE DYSTROPHY.

Although no very extensive research has been made regarding the condition of bones other than the aural capsule in cases of otosclerosis the consensus of opinion is that abnormalities are limited to the capsule of the ear. It is true that typical otosclerosis has been observed in cases of osteogenesis imperfecta (osteopsathyrosis, brittle bone, fragilitas ossium), and a causal relationship has been thought of, but as yet we have no proof of a common etiology. In a personal communication, Dr. Samuel J. Kopetzky stated that he had found bone abnormalities in otosclerotics, elsewhere than in the aural capsule. One wonders if these cases were true otosclerosis or cases of osteodystrophia fibrosa in which aural capsule changes similar to otosclerosis are seen as part of a generalized bone dystrophy. Certain developmental anomalies resulting in congenital deafness have been accompanied by typical otosclerosis. Here again a common etiology has been suggested but never proven.

DEVELOPMENT OF THE AURAL CAPSULE FROM MESENCHYME TO CARTILAGE.

When in the embryo gastrulation occurs our interest as otologists begins, for the mesoderm, last of the three germinal layers to appear, furnishes the pluripotential mesenchyme cells which will build the aural capsule in which otosclerosis develops. By the tenth day[4] the primitive groove, from which the mesoderm is derived, has receded caudalward and cephalad the neural groove has formed. The edges of the neural groove approach each other and by the sixteenth day fuse to form the neural canal. The neural canal soon expands into the brain and by the twentieth day two thickenings in the wall of the myeloncephalon have appeared as the auditory placodes. Opposite each placode the brain sends out neural tissue as the facio-acoustic ganglion. The myeloncephalon becomes the medulla, from which project the ramus cochlearis and the ramus vestibularis.

By the twenty-fourth day the auditory placode has invaginated to form the foveola labyrinthica. When the edges of the foveola

have fused and the invagination has severed its connection with the ectoderm, it becomes (on the twenty-eighth day) the auditory vesicle. The vesicle is surrounded by mesenchymal cells which immediately condense to establish the first step in capsule formation (Fig. 15). In those who are to be afflicted with otosclerosis or who may transmit the condition, these mesenchymal elements, we believe, contain the genes of regression. By the thirty-first day the mesenchymal cells for the capsule can be readily distinguished from the remaining cephalic mesenchyme. The vesicle shows a dorsomesial projection, the recessus labyrinthi (future ductus endolymphaticus). By the forty-fifth day the capsule shows beginning precartilage formation. Semicircular canals have formed from two evaginations of the vesicle, which is constricting into utriculus and sacculus. A saccular evagination, the ductus cochlearis, reminds us of the lagena cochleæ of fishes. The capsule is now very similar to that of the lowest vertebrates, having no oval window. Near the glossopharyngeal nerve is an area of lesser condensation, indicating the round window.

At forty-five days the capsule of condensed mesenchyme is surrounded by a plexus of blood vessels (perichondrium has not yet formed). The nuclei of the mesenchymal cells at the periphery are seen to grow larger and the cells to separate. Of the many protoplasmic processes, some grow more prominent while others disappear. The large ones curve and coalesce to enclose the cells in lacunæ. Between the separating cells an opaque substance forms and takes the hematoxylin stain very lightly. This substance (matrix) shows fibrillar network for a while and then becomes quite homogeneous with the ordinary stains, as the precartilage (Figs. 13, 14) changes into true cartilage. When the auditory vesicle is shut off from ectoderm it shows on its external surface a group of syncytial cells which during the precartilage stage are augmented by a conversion of the precartilage elements adjoining the membranous labyrinth into a syncytial reticulum (Fig. 14). This reticulum, as we learned in the previous chapter, is resorbed to form the perilymphatic (periotic) spaces; strands of the reticulum remaining about the canals to transmit vessels and to act as supports to the membranous ducts. At about three months, mesenchymal cells surrounding the capsule have dif-

ferentiated into fibroblasts, which arrange themselves in parallel alignment to form the fibrous perichondrium. The outer cells of the syncytial reticulum likewise condense at the precartilage border to form the inner perichondrium.

The perichondrium shows an outer fibrous layer and an inner cellular layer. From the cellular layer are differentiated the chondrogenic osteogenic cells which form cartilage cells and later osteoblasts and osteoclasts. The collagen fibers of the perichondrium are acidophilic; the cartilage cells and matrix are basophilic.

At sixty days the capsule is of precartilage. There is as yet no indication of the oval window. The stapedial anlage is noted as a projection of Reichert's cartilage (cartilage of the second arch) into the tympanum. This anlage, pressing against the capsule, will later form the crura of the stapes. Around each canal is noted a halo (Fig. 14) of syncytial reticulum which will later be resorbed to leave a periotic space.

Gradually the precartilage changes into a darker staining tissue of more definite form: true cartilage. At this time a true perichondrium has formed.

CARTILAGE STAGE TO BONE.

At 2½ months the aural capsule has become cartilaginous except where the oval and round windows, internal meatus and cochlear aqueduct are situated. Over the windows are mesenchymal curtains. The round window curtain differentiates into fibroblasts and then into connective tissue to finally become the membrana tympani secundaria. In the case of the oval window curtain the process is more complicated. The stapedial ring is in apposition to the oval window curtain which is composed of mesenchyme cells. At this stage the oval window is analogous to that of the clupea harengus, acantheus vulgaris and raja clavata, namely, membrane covered.

At three months the capsule is of adult cartilage staining deeply with hematoxylin. The stapedial ring has flattened on its median surface to accommodate itself to the curtain, now of precartilage. Reichert's cartilage shows an atrophy separation from the ring. The annular ligament, first appearing phylogenetically in the

menopoma alleghanniense, shows at this period, in mammalians, distinct differentiation. The cisterna perilymphatica, median to the curtain, is rapidly clearing of syncytial reticulum.

At four months the ring has fused with the curtain, which has changed to cartilage and may now be designated as the footplate of the stapes. The annular ligament is well differentiated. The stapedial artery is still present. Ossification of the capsule is well under way.

Fig. 1 (4½ months) shows ossification which stops abruptly on either side of the oval window where cartilage persists. Frontal section.

In Fig. 2 (4½ months) we visualize, in the center, the stapes in articulation with the long process of the incus. Above the stapes is a center of ossification in the capsule. Below the stapes is the promontory with another center of ossification. The periphery of both centers stains darker because of the presence of perichondrial bone. In addition, the centers are composed of excavated cartilage undergoing rapid ossification. To the median side of the promontory is the beginning of the basal turn of the cochlea. External to the scala tympani is the membrana tympani secundaria covering the round window. On the tympanal side is a mass of liquifying mesenchyme (gallert Gewebe) limited by entoderm. As the mesenchyme is resorbed the pad will become thinner and thinner until finally the entoderm will form the thin outer layer of the secondary tympanic membrane and the lining membrane of the tympanum.

In Fig. 3 (4½ months) the annular ligament region is shown under higher magnification. On either side of the ligament are undifferentiated mesenchyme cells.

In Fig. 4 (4½ months) is shown a frontal section made just anterior to the oval window. From the vestibule on the right, the fissula ante fenestram is seen passing through the capsule to the tympanum on the left. The canal opens into a mass of gallert Gewebe in the middle ear. Below is a center of ossification. Note the heavy perichondrial bone on the surface, showing areas of rupture.

In Fig. 5 (4½ months) the fissula is seen passing through the capsule in relation to the processus cochleariformis. Below the

fissula is a center of ossification. The lower left corner of the photomicrograph shows part of the parotid gland.

Posterior to the oval window is an outpouching (Fig. 6) of the capsule, the fossula post fenestram. Under higher magnification (Fig. 7) the fossula is seen to be lined with fibroblasts and young connective tissue. The structure rarely passes to the tympanum. Its origin is a complete mystery. Note the invasion of the capsule by the cells lining the fossula as if attempting to force a passageway into the tympanum.

THE HISTOLOGIC PICTURE OF CAPSULAR OSSIFICATION.

In order to understand the histopathologic picture of otosclerosis and the pathogenesis of the condition, a very complete understanding of the whole process of capsular ossification is indispensable, from its onset just before the fourth fetal month to its completion. First let us visualize a cartilage model covered with perichondrium. Within the cartilage model is the membranous labyrinth surrounded by the potential periotic spaces containing syncytial reticulum and lined with perichondrium.

In certain areas, at varying times, the process of ossification is initiated by the deposit of small quantities of calcium in the cartilage matrix. This causes the area to take the hematoxylin more deeply (Fig. 4) and, upon being cut, to give a gritty sensation. Next, the cartilage cells swell, thereby distending the lacunæ. The cells and their nuclei later become pyknotic, but the lacunæ remain enlarged. About this time ruptures of the perichondrium occur. Through the ruptures, now pass into the cartilage model, blood vessels preceded by histiocytes and accompanied by osteogenic cells (Fig. 8). These vessels are directly differentiated in the surrounding perichondrium and have but a single layer of endothelial cells. They are to become the vessels of the Haversian systems. Excavation of the cartilage model ensues (Fig. 9). Some of the lacunæ are invaded by osteoblasts, the cartilage cells succumbing (Fig. 10). Calcium is deposited about these osteoblasts, with the result that osseous structures of cartilage shape are formed. Some cartilage cells are said to persist as such into adult life. Pink-purple staining osteoid now forms about the persisting lacunæ (Fig. 12). On this osteoid, rows of osteoblasts

lay down lamellæ of bone. Meanwhile fiber bone is forming from perichondrium. This perichondrial bone will later be replaced in part by Haversian system invasion. Around the periotic spaces a band of fiber bone is formed from syncytial reticulum, the endosteal layer. Between the two is the enchondral labyrinth. Enchondral bone continues to form at the expense of the primary marrow until the latter is narrowed down to the Haversian canals and adult marrow spaces.

In Fig. 11 we note a heavy calcium deposit in the osteoblasts which have invaded the lacunæ and around the lacunæ osteoid has formed. Above and below the nest of lacunæ are primary marrow spaces with osteoblasts in epithelioid alignment (seen below), laying down lamellar enchondral bone. Fig. 12 shows the same process.

Having arrived at the stage of lacunar calcification with its surrounding osteoid, fringed with osteoblasts, our next step is the study of matrix formation by these osteoblasts. According to Ham, the osteoblasts now secrete a red staining fibrillar matrix which anchors to the osteoid, enclosing the osteoblasts as osteocytes. The fibrils arrange themselves in bundles as the enchondral bone lamellæ.

In Fig. 18 (age 7 months, fetal), we have an illustration of enchondral bone formation. The oval ring of enchondral lamellar bone surrounds a mass of primary marrow. Lining the marrow cavity are a number of large dark staining osteoblasts. Soon this marrow cavity will narrow down to an Haversian canal. Surrounding the oval ring is a stellate configuration of enchondral bone enclosing large, round and oval calcified cartilage lacunæ. Note the osteocytes within the cartilage rests. Some of these rests are more or less uniformly gray (complete calcification), while some have white centers (incomplete calcification). Around the cartilage rests light gray or white (in the specimen, pink-purple) osteoid is seen. Osteoid is incompletely calcified bone. The osteocytes with their clear protoplasm are dotted throughout the enchondral bone in fairly regular arrangement. The lamellæ of the enchondral bone are bundles of fibrils formed by the osteocytes. Surrounding the stellate enchondral trabeculæ is again primary

marrow and along the external border are large osteoblasts, particularly well seen to the left of number 1.

In Fig. 19 from the same specimen (7 months fetal) is seen a bar of normal enchondral bone enclosing ossified cartilage rests. Between the cartilage rests is osteoid. Above and below is primary marrow and along the trabecula of bone are osteoblasts in epithelioid alignment. The osteocytes are regularly arranged between and in the lamellæ.

Even as early as 4½ months, fetal age, we have already learned that perichondrial bone is well developed but not yet as solid as we now visualize it at 7 months (Fig. 20). Note the clean, solid lamellæ. To the left of the bar of periosteal bone is primary marrow surrounding a trabecula of enchondral bone. Within the latter are cartilage rests and osteoid.

In Fig. 21 (same specimen) we see a low power view of all the capsular layers. Above is dura. Beneath the dura is the broad layer of perichondrial bone, overlying the superior canal. Immediately bordering the canal is a layer of endosteal bone. Between the two we see the broad expanse of enchondral bone trabeculæ surrounded by primary red marrow. In some places the enchondral bone extends to the dura.

POSTNATAL CHANGES IN THE CAPSULE.

As time goes on, the enchondral trabeculæ grow thicker and thicker, thereby diminishing the primary marrow areas.

In Fig. 22, from a 2-months-old child, we note the marked increase in the bars of enchondral bone. Although an occasional large marrow space is still present, most of these areas have diminished in size. Cartilage rests are very numerous. Bordering the cochlea is the solid layer of endosteal bone. Under higher magnification (Fig. 23), from the same 2-months-old specimen, the endosteal bone is better seen. Note the smallness of the marrow spaces as compared to the 7-months fetal specimen. Although the enchondral bone has increased markedly in amount, it does not have the appearance of solidity that we shall see in older specimens. In this picture is nothing that can be definitely identified as an Haversian canal. In Fig. 24, from the same 2-months-old specimen, we see large bars of enchondral bone enclosing car-

tilage rests and surrounded by red marrow. At (6) is endosteal bone sharply separated from enchondral bone by a dark line of cementum. A single Haversian system (3) has made its appearance.

The same specimen (2-months-old child) is seen in Fig. 25 under much higher magnification. We are first impressed by the compactness of the enchondral bone; although compact, it lacks the hardness of appearance which will be noted later.

In the center of the mass of cartilage rests is noted a most interesting phenomenon. A group of calcified cartilage discs are being incorporated into enchondral bone proper (just above the number 1). At (1) is the usual intercartilaginous osteoid. Fig. 26 is again a view of the same 2-months-old specimen, in which we see the beautifully regular arrangement of lamellæ and osteocytes in the periosteal bone.

From this time on, the changes are chiefly an increasing density of all bone structures, the completion of numerous primary Haversian units, partial displacement of primary perichondrial bone by Haversian systems, degeneration of primary Haversian units and formation of new ones and the laying down of ground plates between the Haversian systems, including the formation of fine fibered lamellarless bone.

At 13 months (Fig. 27) the periosteal bone is firm in appearance, and the cartilage rests are so completely ossified as to appear not unlike the surrounding enchondral bone. Marrow spaces are small and infrequently encountered as compared to earlier stages. In Fig. 28 (same specimen, 13 months) we see at a glance how compact the capsule has become. Throughout are well developed primary Haversian systems, seen in cross and longitudinal section. The Haversian canals are visualized either as black lines, straight and branching, or on cross section as circles. Some of these systems will degenerate and give place to firmer secondary systems. The endosteal bone shows Haversian canals crossing it from the enchondral layer. Note how few marrow spaces are left.

In Fig. 29 (13 months), from the same specimen, we see periosteal bone with well formed Haversian systems in cross and longitudinal section. Some of the Haversian canals pass from the periosteal bone into the enchondral bone on the left. In the latter

are well formed Haversian systems. The lower one (1) is surrounded by cartilage rests. Only two marrow spaces are present in this picture.

We have seen, in the 4½ months fetal sections, the narrow fissula ante fenestram traversing the cartilage model from vestibule to tympanum. In Fig. 30 (13 months infant) this structure has grown to much larger proportions and can now be studied in detail. From its beginning in the cisterna perilymphatica just anterior to the stapes we follow it anteriorly until it disappears from view in the enchondral bone. A series of sections would show its course through to the tympanum. In Fig. 31 the fissula is shown in the same section under higher magnification. We now note the cartilaginous lining, which is continuous with the cartilage covering of the capsule at the oval window. Under still higher magnification (Fig. 32) the cartilage walls are visualized in detail and we begin to recognize certain cellular elements in the lumen. It is interesting that the cartilage lining seems to project jaggedly into the lumen without a definite protective perichondrium. The same specimen, under still higher magnification (Fig. 33) shows a wall of unossified cartilage elements. In the lumen may be clearly made out (if one uses a magnifying glass) undifferentiated mesenchymal cells, histiocytes, osteogenic cells, fibroblasts, etc.

In Fig. 34 is a view of the cartilage covered capsule segment with the attached annular ligament (anterior portion). Near the cartilage cells to which the annular ligament is attached one sees undifferentiated mesenchymal elements.

Fig. 35 shows the aqueductus cochleæ passing from the scala tympani. The specimen is from a 2-year-old child.

In Figs. 36 and 37 are shown the posterior and anterior portions, respectively, of the annular ligament from a 13-months-old child. Both the stapes and the capsule segments are covered with a broad layer of cartilage. Undifferentiated mesenchymal cells are noted at the attachment of the annular ligament both to the stapes and to the capsule.

In Fig. 38 (13-months-old child), the perichondrial bone bordering the tympanum is seen. The absence of adult compactness is noticeable.

In Fig. 39, from the same specimen, an Haversian canal is viewed under high magnification. The bone surrounding the canal is not so firm in appearance as that to be seen in the adult sections to follow.

At two years the aural capsule is said to be complete, although certain bone degenerations and reformations occur after this period. Also in old age, sclerosis and rarefication are observed.

If it were possible to film the development of the aural capsule from condensation of mesenchyme to adult bone a most interesting progression and retrogression, with later construction and destruction, would pass before our eyes: mesenchyme into precartilage, precartilage back to syncytial reticulum, precartilage into cartilage, resorption of syncytial reticulum, formation of perichondrium, deposit of calcium in cartilage, swelling of lacunæ, ruptures in perichondrium, excavation of cartilage by invading vessels, ossification of cartilage rests, secretion of osteoid about these cartilage rests, alignment of osteoblasts, building of lamellar bone about the osteoid, transformation of syncytial reticulum into endosteal bone, building of perichondrial bone by osteogenic cells from perichondrium, narrowing down of primary marrow by lamellæ of primary bone to form the Haversian canals. In the 13-months sections all this has been completed, but the capsule of the ear is not through with changes. Just as elsewhere in the osseous system, some of the bone probably dissolves away to give place to new forms. The regular, well formed lamellæ of perichondral bone degenerate and new, firmer ones form. Haversian systems encroach upon the external layers of the capsule. In the enchondral division primary Haversian systems probably disappear and are replaced by new and stronger ones. Most of the ossified cartilage rests seem to remain unchanged: some, as we have seen, are incorporated into lamellar bone. Just what happens to the osteoid between the discs of cartilage is not known. In a 7-months fetus this osteoid stains pinkish-purple. In the adult, the cartilage rests seem to be suspended in air, but when carefully examined under high power we find an almost colorless, slightly opaque substance cementing them together. Just what chemical change has occurred in the osteoid, just how hard it is, etc., the writer does not know.

THE ADULT CAPSULE.

In an adult capsule the Haversian bone lamellæ take a light purple stain in many H-E sections. The bone appears extremely hard and regular. We see it in circles and gracefully winding ribbons. Under oil immersion the bone cells show numerous protoplasmic processes which pass into the canaliculi leading to the lumen of the Haversian units. The bone cells in the Haversian lamellæ show deep staining nuclei for the most part and appear quite vital. A few nuclei take much less stain. In addition to the forms of bone already described there is in the adult capsule a lamellarless bone found between the Haversian systems. This tissue, seen in Fig. 41, gives the impression of porosity. Under high magnification (Fig. 42) this bone shows no lamellæ, but gives, rather, a fine-fibered impression. The porous areas are mostly degenerated bone cells showing no nuclei. Some of the areas seem to be empty spaces. One gets the impression that this bone is the end stage; that no further alterations will occur unless it be a senile rarification. Presumably this lamellarless bone is the fine-fibered bone first described by Max Meyer. Its manner of origin and its exact time of appearance is unknown to the writer. Possibly it represents degenerated Haversian units.

In Fig. 40 from an adult temporal bone we immediately get the feel of solidity in the Haversian systems and even in the numerous ossified cartilage rests. At (1) is the endosteal bone bordering the cochlea, while at (4) we see the extremely dense and wide layer of periosteal bone. The dark bands and circles throughout the enchondral capsule are Haversian canals under various types of sectioning. To the left of (1) is an area of fine-fibered, lamellarless, porous-appearing bone.

Under higher magnification (Fig. 41), in a different adult temporal bone, is seen the lamellarless bone at (4). The porosities are clearly noted. The clear areas around the cartilage rests are not spaces but a translucent end product of osteoid. At (5) is the firm lamellar Haversian bone; (6) is a cross section of an Haversian system.

In Fig. 42, again from a different adult temporal bone, we see under still higher magnification, the various elements already

described. At (1) is fine-fibered lamellarless bone. The calcified cartilage elements are here seen embedded in the modified osteoid. Around each disc is a clear area which seems to be due to retraction of the matrix. Above the cartilage rests is lamellar enchondral bone with an Haversian canal cut longtitudinally.

In Fig. 43, a section from another adult capsule, we see under still higher magnification an area of cartilage rests surrounded by matrix which here appears as clear space. At (1) is an area of lamellarless bone. Above and below the cartilage rests is lamellar enchondral bone.

We have now completed our study of the development of the aural capsule and are in a position to appreciate the various phenomena comprising the pathogenesis of otosclerosis.

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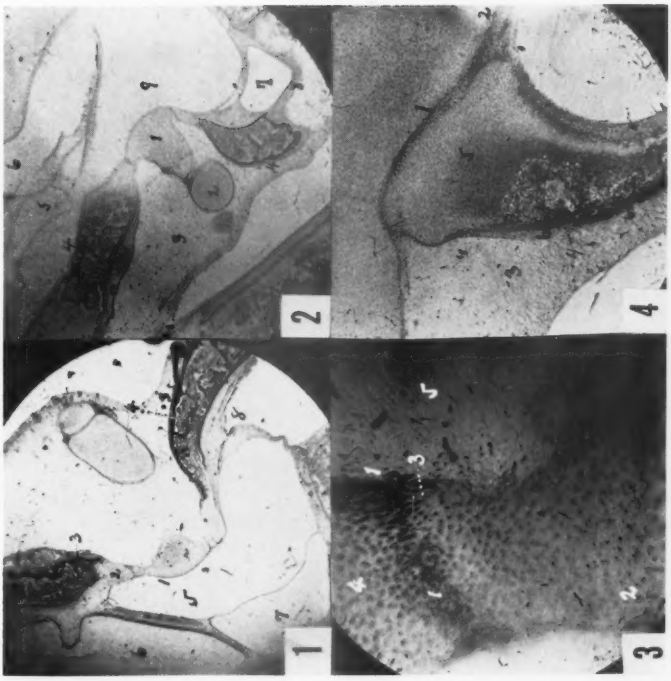
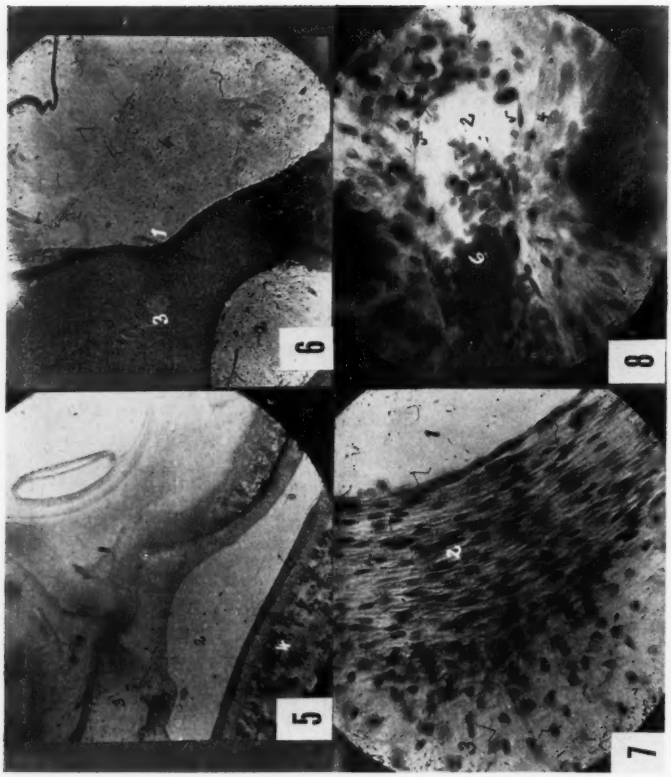


PLATE ONE *Use a magnifying glass.*



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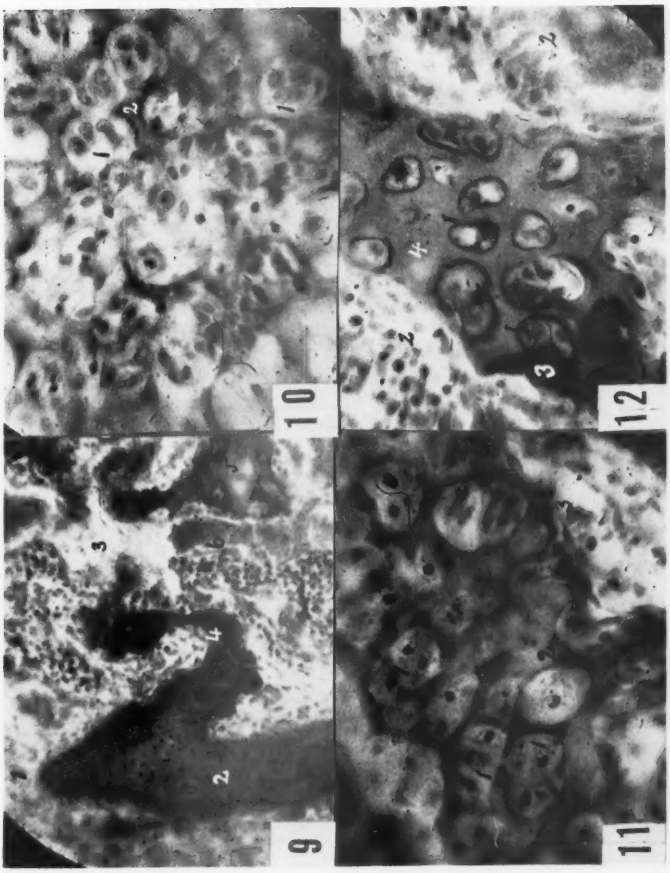

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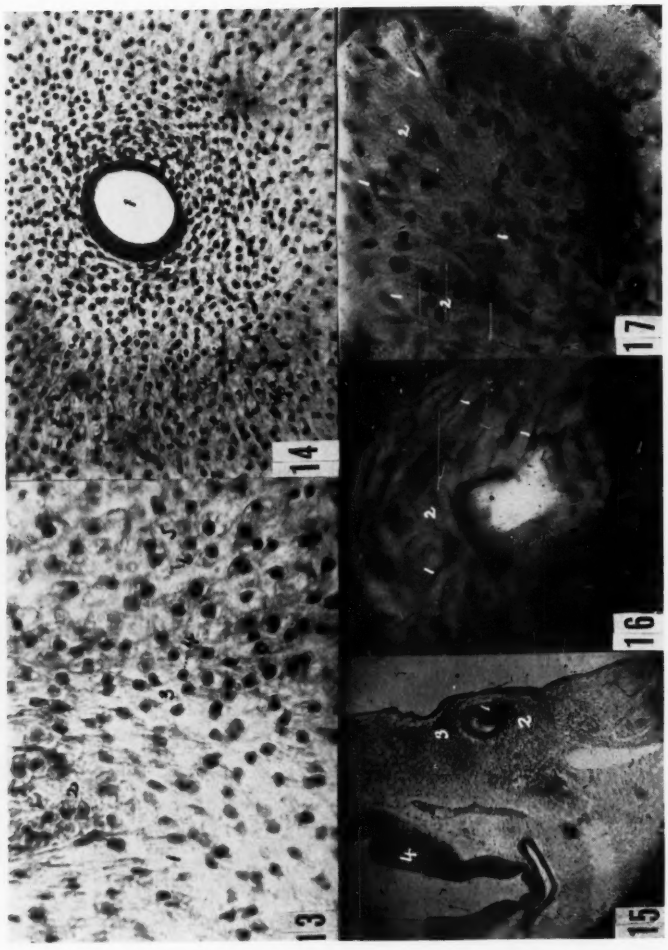

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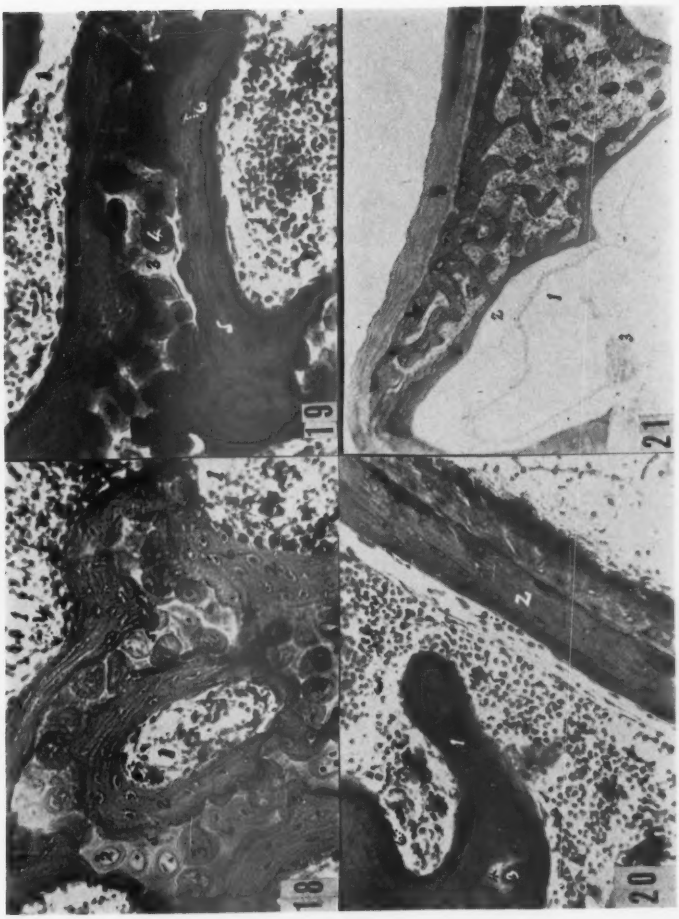

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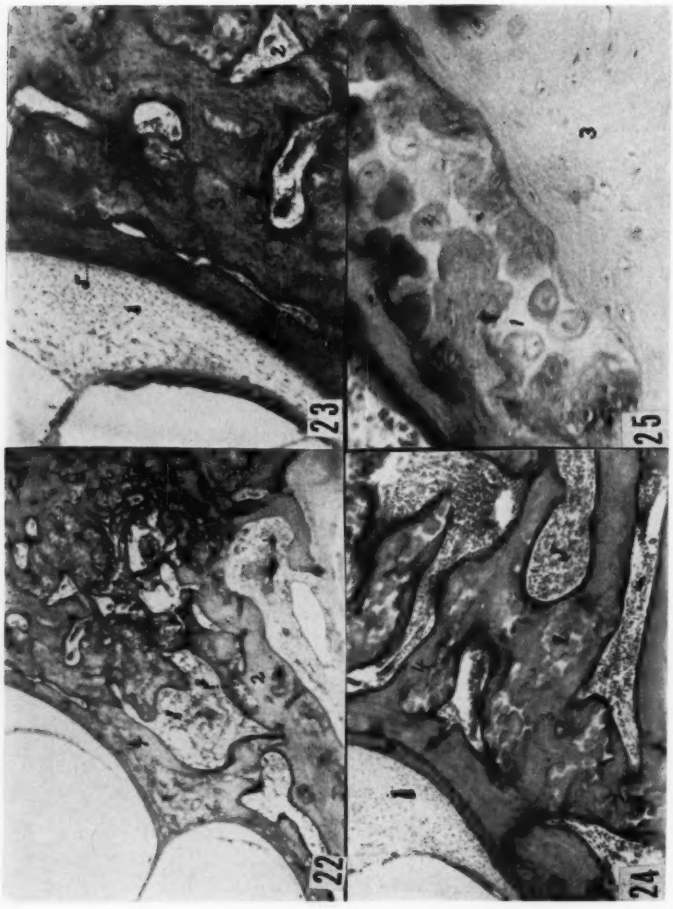

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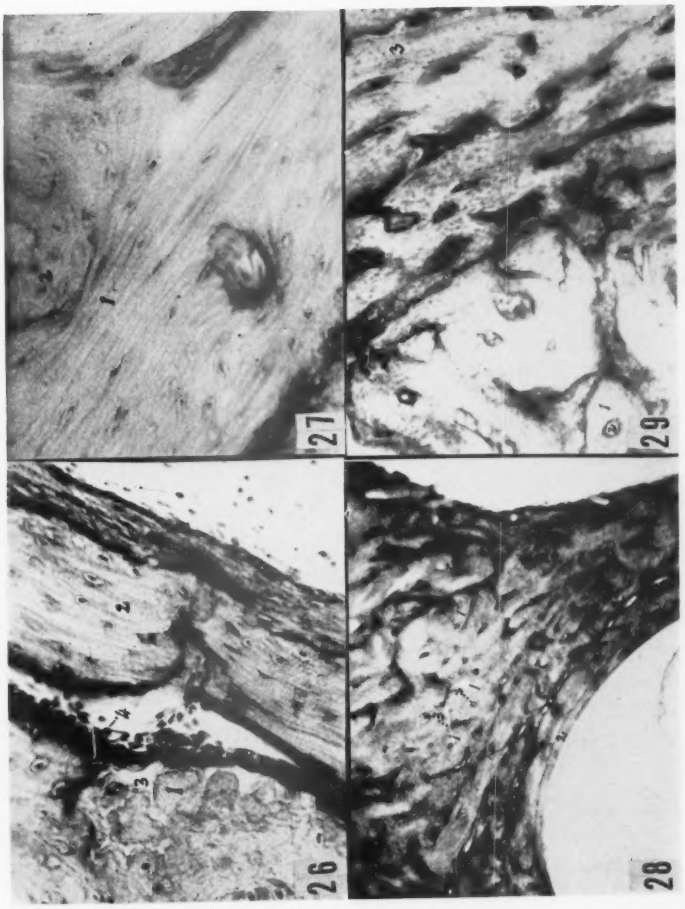

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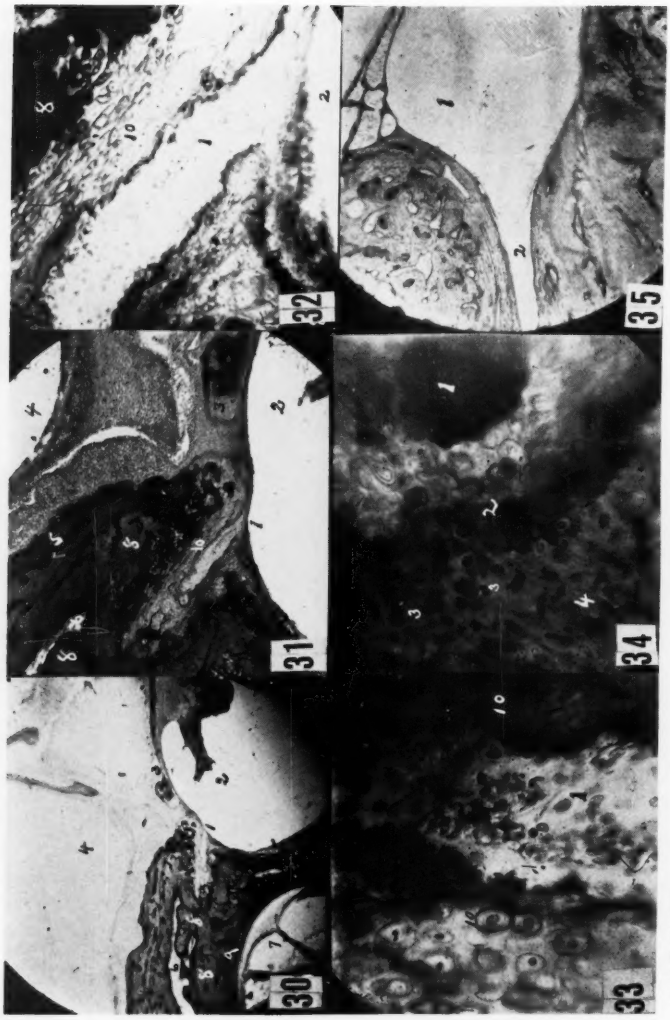

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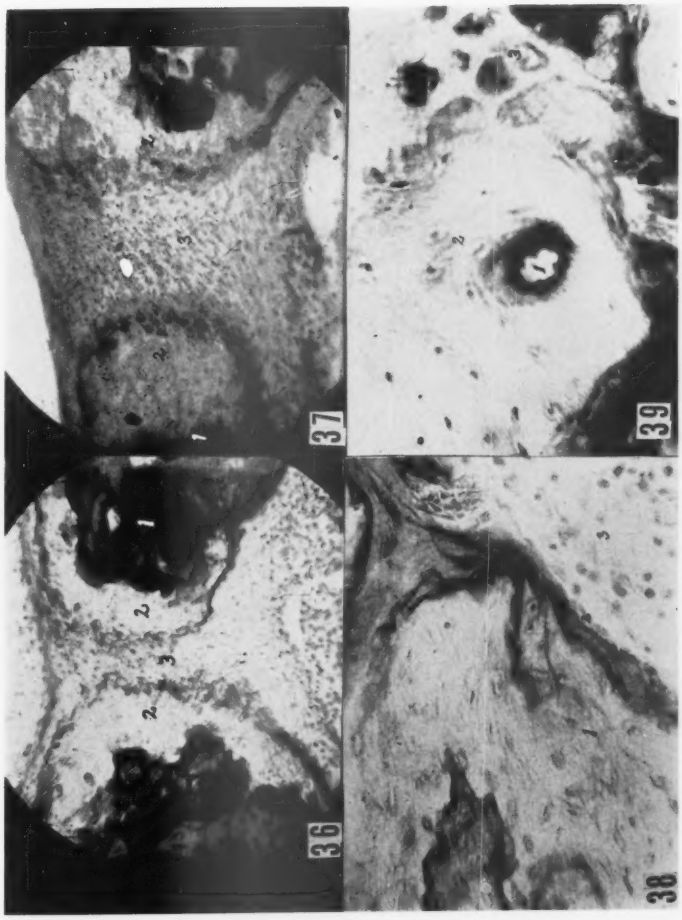

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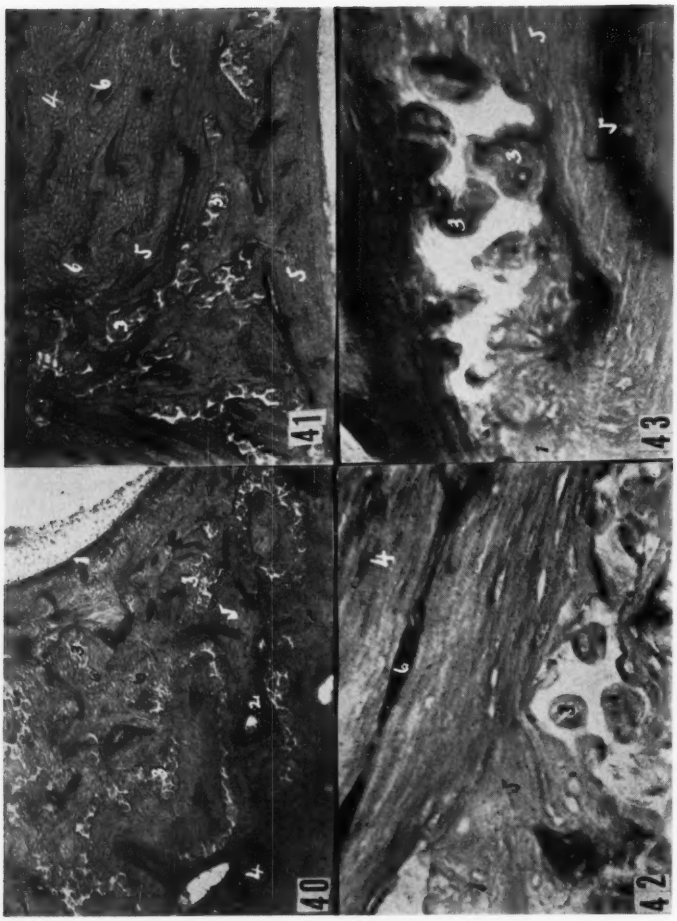

PLATE TEN

Use a magnifying glass.

LEGENDS FOR ILLUSTRATIONS.

PLATE ONE.

Fig. 1. Human (age 4¼ months)—*1*. Footplate of stapes. *2*. Cartilaginous capsule. *3*. Ossification center. *4*. Perichondrial rupture. *5*. Cisterna. *6*. Utriculus. *7*. Sacculus. *8*. Cochlea.

Fig. 2. Human (age 4½ months)—*1*. Stapes. *2*. Incus. *3*. Gallert Gewebe (mesenchyme). *4*. Center of ossification. *5*. Ampulla of horizontal canal. *6*. Anterior vertical canal. *7*. Scala tympani. *8*. Membrana tympani secundaria. *9*. Cisterna.

Fig. 3. Human (age 4½ months)—*1*. Annular ligament. *2*. Footplate of stapes. *3*. Undifferentiated mesenchyme cells. *4*. Caspule. *5*. Gallert Gewebe in tympanum.

Fig. 4. Human (age 4½ months)—*1*. Fissula ante fenestram. *2*. Cisterna. *3*. Gallert Gewebe in tympanum. *4*. Center of ossification. *5*. Matrix showing calcium deposit. *6*. Perichondrial ruptures.

PLATE TWO.

Fig. 5. Human (age 4½ months)—*1*. Fissula ante fenestram. *2*. Tympanum. *3*. Processus cochlearformis. *4*. Parotid gland.

Fig. 6. Human (age 4½ months)—*1*. Fossula post fenestram. *2*. Tympanum. *3*. Caspule. *4*. Vestibule.

Fig. 7. Human (age 4½ months)—Higher magnification of Fig. 6. *1*. Cisterna. *2*. Fibroblasts lining fossula. *3*. Cartilaginous capsule.

Fig. 8. Human (age 4½ months)—*1*. Perichondrial rupture. *2*. Capillary. *3*. Histiocytes. *4*. Osteogenic cell. *5*. Endothelium. *6*. Erythrocytes.

PLATE THREE.

Fig. 9. Human (age 4½ months)—*1*. Perichondrial rupture. *2*. Perichondrial bone. *3*. Excavated cartilage model. *4*. Calcified cartilage lacuna. *5*. Osteoid. *6*. Osteoblasts in epithelial alignment.

Fig. 10. Human (age 4½ months)—*1*. Cartilage lacunæ showing osteoblasts in place of cartilage cells.

Fig. 11. Human (age 4½ months)—*1*. Cartilage lacunæ with calcifying osteoblasts. *2*. Osteoid undergoing calcification. *3*. Primary marrow space with osteoblasts in epithelial alignment.

Fig. 12. Human (age 4½ months)—*1*. Calcifying cartilage lacunæ. *2*. Primary marrow space. *3*. Osteoid with calcium being deposited. *4*. Osteoid.

PLATE FOUR.

Fig. 13. Two months' old human embryo—Frontal section through the outer portion of aural capsule. *1*. Preperichondrial layer of mesenchyme. *2*. Engorged vessel in preperichondrium. *3*. Osteogenic layer of preperichondrium. *4*. Osteogenic cell (chondroblast) differentiating from preperichondrium. *5*. Matrix forming in capsule (precartilage stage). *6*. Protoplasmic process of chondroblast forming lacuna.

Fig. 14. Two months old human embryo—Frontal section through a semicircular canal. *1*. Canal. *2*. Syncytial reticulum formed from precartilage. *3*. Precartilage area. *4*. Protoplasmic process of chondroblast coalescing with another process to form a lacuna. *5*. Syncytial reticulum closed off originally with the auditory vesicle.

Fig. 15. Twenty-eight day human embryo—Frontal section. *1*. Auditory vesicle. *2*. Condensed mesenchyme (beginning of capsule formation). *3*. Facio-acoustic ganglion. *4*. Myelencephalon.

Fig. 16. Adult human aural capsule—A marrow space. *1*. Undifferentiated pluripotential mesenchyme cells. *2*. Fibrous reticulum.

Fig. 17. Thirteen months old human—Lumen of fissula ante fenestram. *1*. Undifferentiated pluripotential mesenchyme cells. *2*. Histiocytes. Also present: fibroblasts, chondroblasts.

PLATE FIVE.

Fig. 18. Seven months human fetus—Frontal section through aural capsule. *1*. Primary marrow. *2*. Lamellar enchondral bone. *3*. Calcified cartilage rest. *4*. Osteoblast. *5*. Osteoid. *6*. Osteocyte.

Fig. 19. Seven months human fetus—Frontal section through capsule. *1*. Primary marrow. *2*. Osteoblasts. *3*. Osteoid. *4*. Calcified cartilage rest. *5*. Lamellar enchondral bone. *6*. Osteocyte.

Fig. 20. Seven months human fetus—Frontal section through aural capsule. *1*. Enchondral bone. *2*. Perichondrial lamellar bone. *3*. Cartilage rest. *4*. Osteoid. *5*. Primary marrow. *6*. Osteoblasts.

Fig. 21. Seven months human fetus—Frontal section. *1*. Ampulla of superior canal. *2*. Perilymphatic space. *3*. Crista. *4*. Primary marrow. *5*. Endosteal bone. *6*. Periosteal bone. *7*. Enchondral bone. *8*. Dura.

PLATE SIX.

Fig. 22. Two months old child—Aural capsule. *1.* Primary marrow. *2.* Enchondral bone. *3.* Cartilage rests. *4.* Endosteal bone.

Fig. 23. Two months old child—Aural capsule. *1.* Ligamentum spiralis. *2.* Marrow *3.* Enchondral bone. *4.* Cartilage rests. *5.* Endosteal bone.

Fig. 24. Two months old child—Aural capsule. *1.* Ligamentum spiralis. *2.* Cartilage rests. *3.* Haversian system. *4.* Enchondral bone. *5.* Marrow. *6.* Endosteal bone.

Fig. 25. Two months old child—Aural capsule. *1.* Osteoid. *2.* Calcified cartilage. *3.* Enchondral lamellar bone.

PLATE SEVEN.

Fig. 26. Two months old child—Aural capsule. *1.* Calcified cartilage. *2.* Lamellar perichondrial bone. *3.* Osteoid. *4.* Osteoblasts.

Fig. 27. Thirteen months old child—Aural capsule. *1.* Lamellar perichondrial bone. *2.* Cartilage rests.

Fig. 28. Thirteen months old child—Aural capsule. *1.* Haversian system. *2.* Endosteal bone. *3.* Haversian canal.

Fig. 29. Thirteen months old child—Aural capsule. *1.* Haversian system of lamellar bone. *2.* Haversian canal. *3.* Perichondrial bone.

PLATE EIGHT.

Fig. 30. Thirteen months old child—*1.* Vestibular entrance to fissula ante fenestram. *2.* Cisterna perilymphatica. *3.* Footplate of stapes. *4.* Tympanum. *5.* Periosteal bone. *6.* Marrow space. *7.* Ductus cochlearis. *8.* Enchondral bone. *9.* Endosteal bone.

Fig. 31. Higher magnification of fissula region—Fig. 30. *1.* Entrance to fissula ante fenestram. *2.* Cisterna. *3.* Stapes. *4.* Tympanum. *5.* Periosteal bone. *6.* Marrow space. *8.* Enchondral bone. *10.* Cartilage lining of fissula ante fenestram.

Fig. 32. Higher magnification of fissula in Fig. 31—*1.* Lumen of fissula ante fenestram. *2.* Cisterna. *8.* Enchondral bone. *10.* Cartilage walls of fissula ante fenestram.

Fig. 33. Higher magnification of fissula in Fig. 32—*1.* Lumen of fissula ante fenestram showing undifferentiated pluripotential mesenchyme cells, histiocytes, fibroblasts, chondroblasts, etc. *10.* Cartilage walls of fissula.

Fig. 34. Thirteen months old child—Annular ligament and capsule segment. *1.* Bone of capsule. *2.* Cartilage covering of capsule. *3.* Mesenchymal cells to left of number. *4.* Annular ligament.

Fig. 35. Two-year-old child—Section through cochlea. *1.* Scala tympani. *2.* Aquaeductus cochleae.

PLATE NINE.

Fig. 36. Thirteen months old child—Section through posterior division of annular ligament. *1.* Footplate of stapes. *2.* Cartilage cover. *3.* Posterior division of annular ligament.

Fig. 37. Anterior division of annular ligament—Same specimen as Fig. 36. *1.* Footplate. *2.* Cartilage cover. *3.* Annular ligament.

Fig. 38. Thirteen months old child—Section through aural capsule. *1.* Periosteal bone. *2.* Periosteum. *3.* Tympanum.

Fig. 39. Thirteen months old child. *1.* Haversian canal. *2.* Lamellar Haversian bone. *3.* Calcified cartilage.

PLATE TEN.

Fig. 40. Adult human. Section through aural capsule—*1.* Endosteal bone. *2.* Haversian bone. *3.* Cartilage rests. *4.* Periosteal bone. *5.* Enchondral bone. *6.* Fine-fibered lamellarless bone.

Fig. 41. Adult human. Section through aural capsule—*3.* Cartilage rests. *4.* Fine-fibered lamellarless bone. *5.* Lamellar bone. *6.* Haversian units.

Fig. 42. Adult human. Section through aural capsule—*1.* Fine-fibered lamellarless bone. *3.* Cartilage rests. *4.* Lamellar Haversian bone. *5.* Fine-fibered bone. *6.* Haversian canal.

Fig. 43. Adult human—Section through aural capsule. *1.* Fine-fibered lamellarless bone. *3.* Cartilage rests. *5.* Lamellar enchondral bone.

XCII.

SUGGESTED MODIFICATIONS OF THE LARYNGO-FISSURE OPERATION.*

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NEW YORK.

INTRODUCTORY.

In reading the history and development of the laryngo-fissure operation, one cannot help having a great feeling of admiration for those who, handicapped by the lack of present means and knowledge in arriving at an early diagnosis, nevertheless persisted in attacking the problem along the present lines.

True, there were periods of pessimism due to poor results, in which the operation fell into disrepute, but nevertheless progress was made, and those real pioneers who had been strong in their condemnation of the procedure, made a complete reversal of their stand when convinced that, after all, there was some merit to the operation.

St. Clair Thomson and Lionel Colledge in their excellent book "Cancer of the Larynx" state:

> "As late as 1897 Bosworth did not think the mention of thyrotomy was necessary in his well-known textbook. But in the years subsequent to 1886, further experience completely altered the opinion of the two chief pioneers, viz., Semon and Butlin. Until that date they had united in condemning thyrotomy as an operation 'which should not be attempted' (Semon) 'in a disease evidently far too deep seated to admit of removal by so slight an operation' (Butlin). By 1894—i. e., eight years after—Semon had, in his own words, 'not merely modified but completely changed his views in regard to thyrotomy'."

While many refinements in technic have been made, the greatest advance is due to the present accurate methods of arriving at an early diagnosis, such as direct laryngoscopy and biopsy.

As this presentation is not essentially concerned in the history of the operation, the reader is referred to the writings of St.

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*Candidate's thesis, American Laryngological, Rhinological and Otological Society, 1932.

From Department of Anatomy of the University and Bellevue Hospital Medical College and Manhattan Eye, Ear and Throat Hospital.

Clair Thomson,[1] St. Clair Thomson and Lionel Colledge,[2] Morell Mackenzie,[3] J. Nolan Mackenzie,[4] not to mention F. Semon, Chevalier Jackson, John E. Mackenty, Gabriel Tucker, Gordon B. New and others.

ANATOMIC CONSIDERATION.

St. Clair Thomson and Lionel Colledge state:

"The most important principle to be observed is an adequate exposure of the tumor before any attempt at excision is begun—".

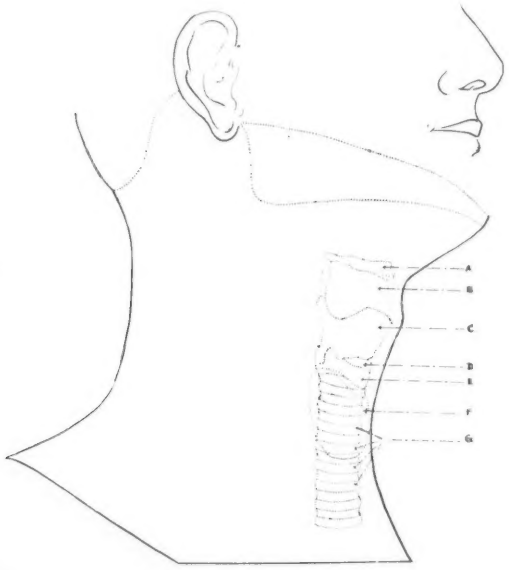

Fig. 1.—Landmarks felt in neck as finger is passed down from chin to suprasternal notch: *A*—Hyoid bone, *B*—Thyrohyoid membrane, *C*—Thyroid cartilage, *D*—Cricothyroid membrane, *E*—Cricoid cartilage, *F*—Thyroid gland, *G*—Tracheal rings.

In order to obtain a good exposure of the interior of the larynx with the present technic, considerable force is necessary in sufficiently retracting the laminæ of the thyroid cartilage; so much, in fact, that not infrequently the cartilage is fractured. Prompted by the desire to overcome this difficulty, an anatomic study of the

region involved was undertaken. The changes to be suggested, based on such a study in forty cadavers, and supplemented by clinical experience, greatly increase the exposure of the interior of the larynx and obviate the necessity for strong retraction.

The anterior part of the neck presents very important and easily identified landmarks. If one passes his finger along the middle

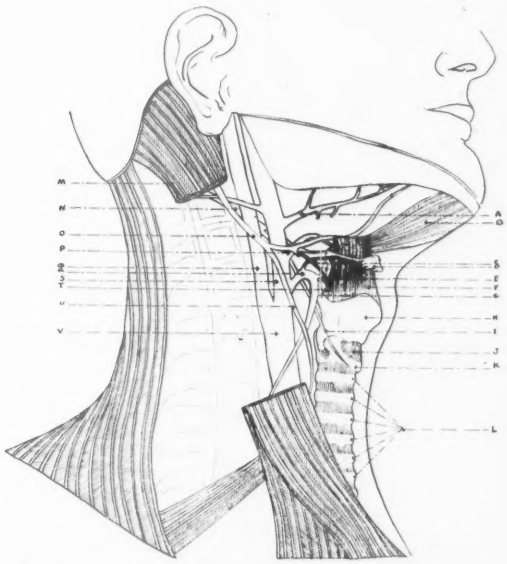

Fig. 2.—Semidiagrammatic drawing of anatomy of neck (side view). *A*—External maxillary artery; *B*—Anterior belly of digastric muscle; *C*—Internal branch of superior laryngeal nerve; *D*—Hyoid bone; *E*—Internal laryngeal artery; *F*—Thyrohyoid membrane; *G*—Greater cornu of thyroid cartilage; *H*—Thyroid cartilage; *I*—External branch of superior laryngeal nerve; *J*—Cricohyoid membrane; *K*—Cricoid cartilage; *L*—Tracheal rings; *M*—Sternomastoid muscle; *N*—Superior laryngeal nerve; *O*—Hypoglossal nerve; *P*—Lingual artery; *Q*—Internal carotid artery; *R*—Superior thyroid artery; *S*—External carotid artery; *T*—Piercing of thyrohyoid membrane by internal laryngeal artery and internal laryngeal nerve; *U*—Descendens hypoglossi; *V*—Common carotid artery.

of the front of the neck from the chin down, he will identify the body of the hyoid bone, the prominent anterior edge of the thyroid cartilage, the cricoid cartilage, the isthmus of the thyroid gland, some of the tracheal rings and finally the upper edge of the manubrium sterni.

Upon palpating these structures more carefully, one finds that the hyoid bone can be easily moved from side to side, and when pressure is applied to say, the left side, the right greater cornu is easily felt. This bone is located at the angle formed by the submental region and the front of the neck with the head neither extended nor flexed.

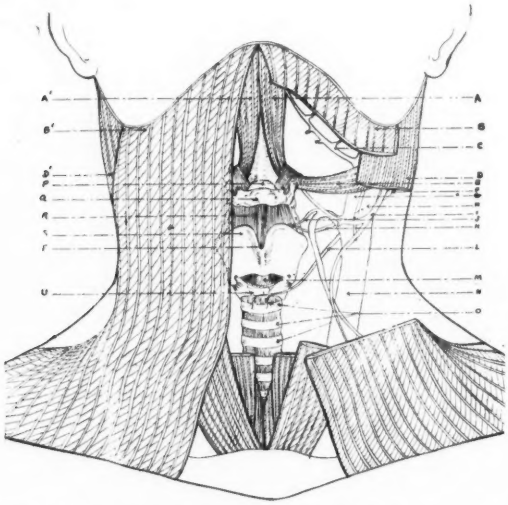

Fig. 3.—Semidiagrammatic drawing of anatomy of neck (front view). *A-A*1—Anterior bellies of digastric muscles; *B-B*1—Platysma muscles; *C*—External maxillary artery; *D-D*1—Sternomastoid muscles; *E*—Posterior belly of digastric muscle; *F*—Superior laryngeal nerve; *G*—External carotid artery; *H*—Internal carotid artery; *I*—Hypoglossal nerve; *J*—Internal laryngeal branch of superior thyroid artery; *K*—Internal branch of superior laryngeal nerve; *L*—External branch of superior laryngeal nerve; *M*—Cricothyroid membrane; *N*—Common carotid artery; *O*—Tracheal rings; *P*—Tip of epiglottis; *Q*—Hyoid bone; *R*—Thyrohyoid membrane; *S*—Thyroid cartilage; *T*—Thyroid notch; *U*—Cricoid cartilage.

Between the lower border of the hyoid bone and the upper border of the thyroid cartilage is a variable space, greater when the head is extended and very much less when flexed. At the upper edge of the anterior border of the thyroid cartilage is a distinct notch. Starting at this point, one can trace the upper border of each wing of this cartilage. It will be found to curve upwards, backwards and 'aterally for a short distance, and then will dip down somewhat but still going posteriorly and laterally.

In not too stout a subject it will be possible to feel the superior cornua. The anterior edge of the thyroid cartilage varies considerably in different subjects. In the male the upper part is very prominent, producing the laryngeal prominence or pomum adami.

Below the lower border of this cartilage and above the upper border of the cricoid there is the cricothyroid space which is much

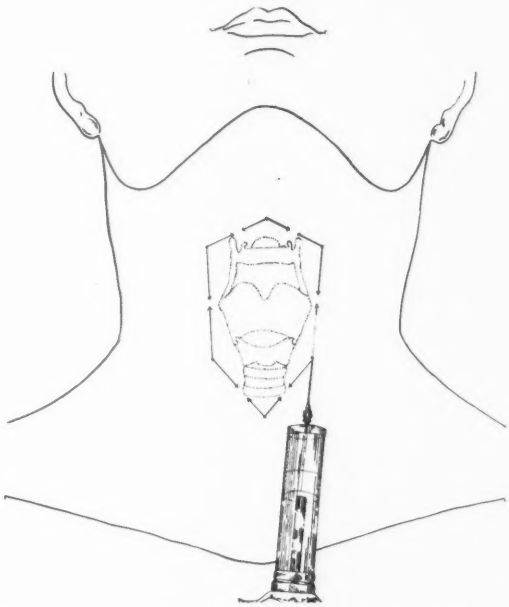

Fig. 4.—Method of field block anesthesia used in laryngo-fissure.

smaller than the thyrohyoid space. This region between the cricoid and the thyroid cartilage is bridged by the cricothyroid membrane, which is crossed superficially by the cricothyroid arteries.

According to Gray, the cricothyroid membrane and the conus elasticus are identical. He states that—

"It consists of three parts, a central triangular portion and two lateral portions. The central part (ligamentum cricothyreoideum medium) is

thick and strong, narrow above and broadening out below. . . . The lateral portions are thinner and lie close under the mucous membrane of the larynx. They extend from the superior border of the cricoid cartilage to the inferior margin of the true vocal cords with which they are continuous."

Cunningham refers to the membrane as "the conus elasticus, which is separable into a median and two lateral parts. The median part is the cricothyroid ligament. Each lateral part . . . terminates in a free thickened border, called the ligamentum vocale." . . .

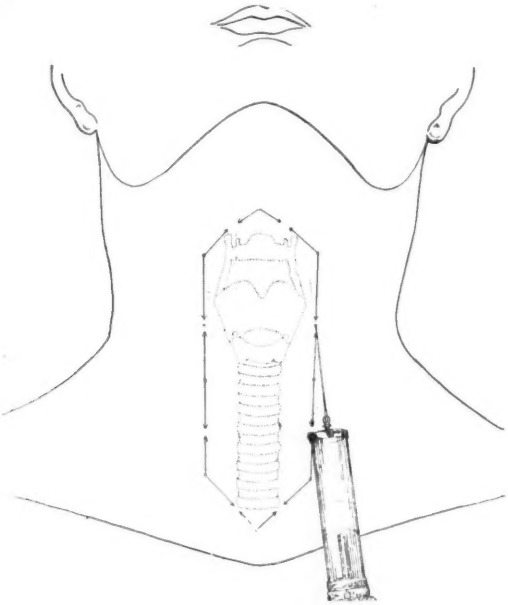

Fig. 5.—Method of field block anesthesia used for combined tracheotomy and laryngo-fissure.

Almost immediately below the lower border of the thyroid cartilage is felt the rounded arch of the cricoid cartilage.

The isthmus of the thyroid gland is felt as a soft, rounded mass, more prominent in some than others, and is usually located anterior to the second, third and fourth tracheal rings.

In thin subjects the tracheal rings below the isthmus of the thyroid gland are easily felt, but in stout ones considerable difficulty may be experienced in palpating them, even with the head

in full extension. It must be remembered that as the trachea approaches the sternum it becomes more deeply situated and may attain a depth of one and one-half inches at the level of the upper border of the sternum. At this point it might be in order to mention that the suprasternal fossa or Burns' space is situated immediately above the upper border of the manubrium sterni.

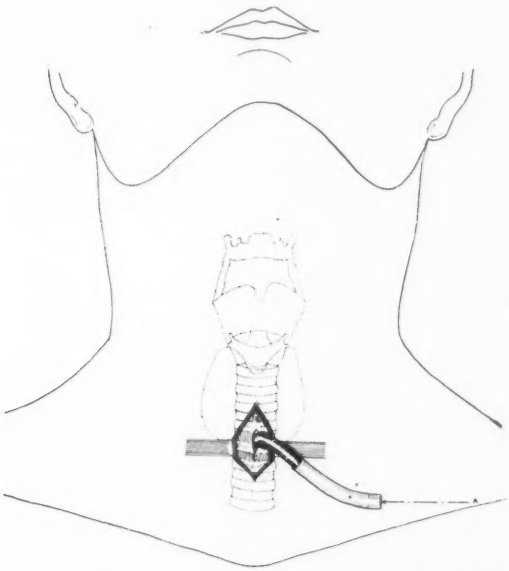

Fig. 6.—Ether tube in tracheotomy wound. Rubber tube *A* is attached to perforated ether can.

Lying superficial to these structures are the skin, superficial fascia and superficial layer of the deep cervical fascia and on each side of the midline two thin layers of muscles, namely, the sternohyoid and omohyoid, and the sternothyroid and thyrohyoid.

This distribution of the muscles produces in the median area a space devoid of muscular covering. In this intermuscular space can be found the cricothyroid arteries, the medial terminal branches of the superior thyroid arteries and the inferior thyroid veins.

Let us return to the thyrohyoid ligament or membrane, for a proper understanding of the anatomy here is important: It is composed of fibrous tissue and extends in one continuous sheet from the upper border of the thyroid cartilage to the posterior surface of the hyoid bone. The posterior edges or borders which extend from the superior cornua of the thyroid cartilage to the tips of the greater cornua of the hyoid are thickened. They may

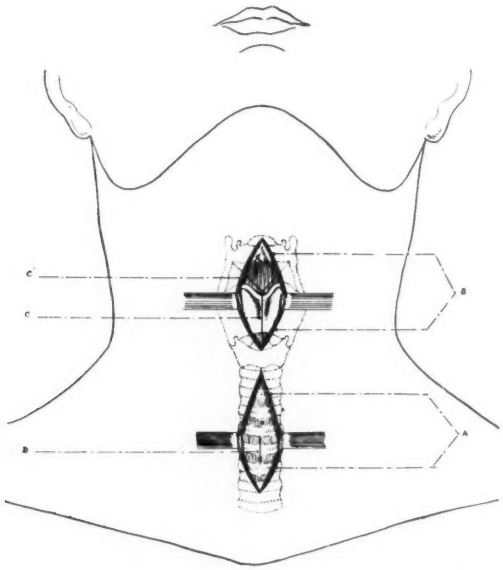

Fig. 7.—Showing two separate incisions for *A*—Tracheotomy and *B*—Laryngo-fissure. *C*-*C*1—shows incision in thyroid cartilage and thyrohyoid membrane; *D*—Incision in tracheal ring.

be artificially dissected to resemble cords (ligamenta thyrohyoidea lateralis), although in fact they are continuous, not only with the rest of the membrane but with the expansion which mingles with the fascia of the neck. Quite often a nodule (cartilage triticea) is found in this lateral thickening.

Piercing this membrane or ligament close to its posterior edge and at a point which bears an almost constant relation to the

uppermost part of the thyroid cartilage, we find a nerve and blood vessels. This nerve which is the internal branch (r. internus) of the superior laryngeal, which in turn is a branch of the vagus nerve, is accompanied by a branch (laryngeal) of the superior thyroid artery and the superior laryngeal vein, a tributary of the superior thyroid vein (v. thyreoidea superior). The internal laryngeal branch of the superior laryngeal nerve passes downward and medially between the middle and inferior constrictor muscles of the pharynx before it pierces the membrane.

It is considered to be essentially a sensory one, although the interarytenoid muscle receives some fibers from it.[5] It supplies the mucous membrane covering the interior of the larynx, the pyriform sinuses and the bases of the tongue and epiglottis (branches of the epiglottic, pharyngeal, descending and communicating). Sensory fibers are also supplied to the mucous membrane lining the lower part of the larynx and to the muscles through the communicating branches which communicate with the terminal filaments of the external laryngeal (r. externus) branch. The point where this nerve (r. internus) pierces the membrane was carefully studied in forty cadavers, and it was found that a straight line drawn across at the level of the uppermost portion of the laminæ proper of the thyroid cartilage to a point within one cm. of the superior cornua of this cartilage would fairly accurately localize the point of entry through the membrane.

Of course there were a few exceptions, but none were important or great enough to make the suggested modification impracticable. The few appended larynges with the measurements show the extremes and average. In none, however, would one be apt to injure either nerve or artery if an incision were made in the thyrohyoid membrane extending from the thyroid notch towards the greater cornu of the hyoid bone.

When this membrane was incised as indicated, it was surprising the view one could obtain of the hyopharynx, pyriform fossæ and the larynx. It practically amounted to a subhyoid pharyngotomy, with the incision so placed, however, that no important structures were injured.

Another relation of some importance is that of the epiglottis to this membrane, the thyroid cartilage and hyoid bone. Fig. 3 shows what this relationship is. There are no other important external anatomic considerations. It now begins to become obvious that any incisions in this membrane, in order to avoid the

nerve and accompanying blood vessels must be so placed that it will start in the midline at the level of the uppermost part of the thyroid cartilage and extend laterally and upward, receding from the upper borders of the laminæ of the thyroid cartilage. An incision so placed, while it may not entirely avoid the stalk of the epiglottis, will only involve it slightly. However, if the incision be started at the notch and carried upward and outward to the level of the upper most part of the thyroid cartilage and then continued as in the first instance the epiglottis will not be injured.

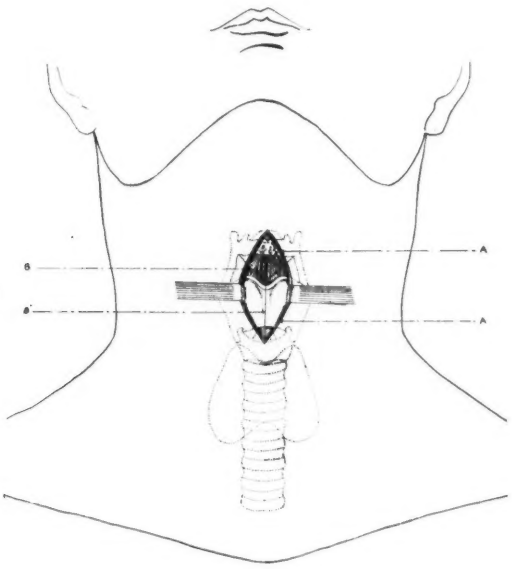

Fig. 8.—*A*-*A*1—Is skin incision for laryngo-fissure operation without preliminary tracheotomy. *B*-*B*1—Is incision in thyroid cartilage and thyrohyoid membrane.

ANESTHESIA—LOCAL.

In performing this operation, the surgeon is presented with the choice of local or general anesthesia. There is no doubt that the technic of inducing local anesthesia has been so perfected as to make it practicable and safe. The indications for this form of

anesthesia will vary according to the viewpoint of the surgeon. Briefly the technic employed may be as follows:

(a) Cocainization of the interior of the larynx with a solution of cocain hcl 10 per cent.

(b) Field block—A solution of novocain, 1 per cent, with a few drops of adrenalin (total amount of adrenalin not to exceed fifteen drops) is used. The extent of the field block will depend on whether a preliminary tracheotomy is intended.

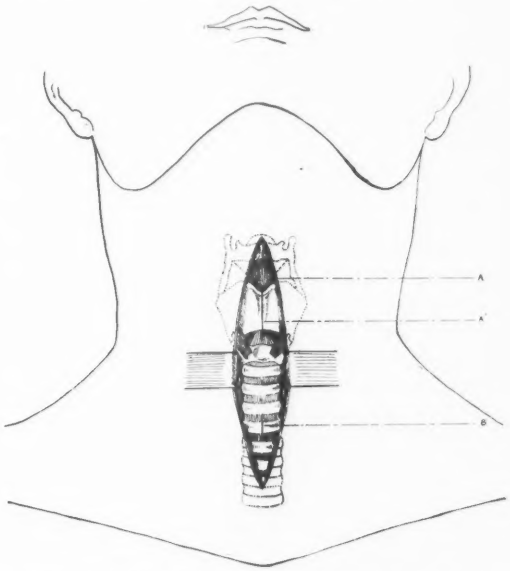

Fig. 9.—Showing exposure of trachea and thyroid cartilage if one continuous incision is used. *A-A*¹—Incision in thyroid cartilage and thyrohyoid membrane. *B*—Incision through tracheal ring for tracheotomy.

Fig. 4 shows the area to be blocked when no tracheotomy is intended. To supplement the intralaryngeal cocainization one may inject both internal laryngeal branches of the superior laryngeal nerve.

Fig. 5 shows the area to be blocked when a preliminary tracheotomy is to be performed. In performing the field block one obtains better results if a few centimeters of the solution are injected beyond the anterior border of the sternomastoid muscle

toward the region where the transverse branch of the cervical plexus emerges.

General.—If general anesthesia is preferred, we may employ (1) intratracheal insufflation anesthesia; (2) anesthesia through a tracheotomy wound, and (3) rectal anesthesia.

The method most frequently used by the writer is that through the tracheotomy wound, the tracheotomy having been previously performed under local anesthesia.

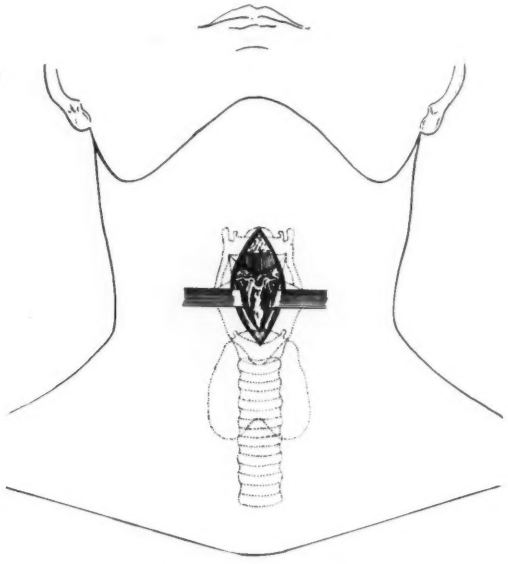

Fig. 10.—Showing degree of exposure of interior of larynx after cutting through thyroid cartilage and thyrohyoid membrane.

Avertin has been used in one case but had to be supplemented with ether.

SKIN INCISIONS—TRACHEOTOMY.

If a preliminary tracheotomy is to be performed, two incisions are employed, rather than a long continuous one. There is no good reason why the incision for the thyrotomy or laryngo-fissure should be done at the same time as, and be continuous with, the

tracheotomy incision, for doing so unnecessarily exposes the thyrotomy wound to an added possibility of infection. Where two incisions have been used, the thyrotomy wound has been seldom infected.

The incisions are, of course, made in the midline, thus reducing bleeding to a minimum.

The incision for the tracheotomy is started at the lower border of the cricoid cartilage and extends to the upper border of the

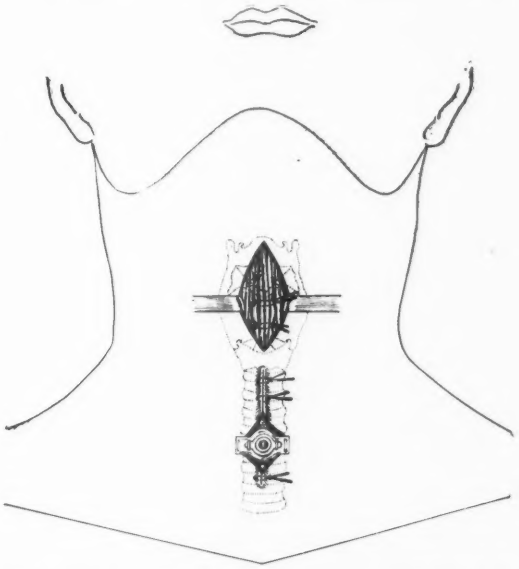

Fig. 11.—Showing closure of tracheotomy wound and partial closure of laryngo-fissure.

manubrium sterni. With the first sweep of the knife the superficial layer of the deep cervical fascia is reached. Should the length of the neck permit, the suprasternal space is avoided, firstly because an infection here may extend into the posterior triangle of the neck, and secondly because as the sternum is reached the trachea becomes deeper and the margin of safety decreases.

The dissection is carried down in the midline to the pretracheal fascia. Under no condition should there be any lateral dissection.

When the pretracheal fascia is reached, the isthmus of the thyroid gland will also have been reached. The writer has made it a habit of opening into the trachea immediately below the lower border of the isthmus of the thyroid gland. Should this isthmus be very broad and extend below the fourth tracheal ring, one may easily push it up if first the fascia is incised over and below it. Rather than cut it, the writer has preferred at times to transfix it with a suture or two to a higher level, exposing one or two additional tracheal rings. The fascia is then incised over the rings to be cut. Should any small veins or the thyroid ima artery be encountered now, unnecessary leakage into the trachea will be avoided by ligating them before the rings are opened. As a rule, two rings suffice. Before opening them, however, a few drops of a weak solution of cocain (5 to 10 per cent) are injected directly into the trachea. Too strong or too much solution should not be used, as this may obliterate nature's protective mechanism. All bleeding must be completely controlled and all cut vessels tied so that the clamps may be removed. The trachea is now opened.

If the operation is to be continued under general anesthesia, a curved metal tube attached to a rubber tube is introduced into the trachea and the anesthesia continued through it. The rubber tube is fixed to the incised skin by means of a suture.

At this stage one may lightly pack the space about the tube to prevent leakage into the trachea.

THYROTOMY OR LARYNGO-FISSURE.

The thyrotomy skin incision is then made. This extends from the body of the hyoid bone to the upper border of the cricoid cartilage and down to the level of the superficial layer of the deep cervical fascia. (In this region the superficial and middle layers of this fascia blend.) At this level the anterior border of the thyroid cartilage lies immediately under this fascial plane. Under no condition should one skeletonize the larynx. The next step is to cut through the cartilage and this can be easily done by means of the Clerf saw. If possible, one should avoid cutting through the anterior commissure at this stage, for should it be necessary to resect more than one cord, one may easily separate

the soft structures from the cartilages on either side of the median line. The larynx is then opened by incising through the soft structures (usually at the anterior commissure). While one may now inspect the interior of the larynx, it will be found that considerable traction is necessary to properly expose the larynx. In order to better visualize the interior of the larynx and to reduce retraction to a minimum, a V-shaped incision is now made in the thyrohyoid membrane, the extent of this incision depending upon

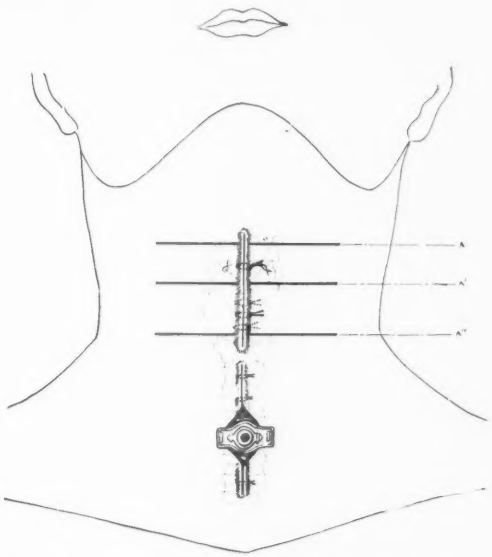

Fig. 12.—Showing closure of tracheotomy wound and laryngo-fissure. A-A1 and A2 are sutures in place to hold gauze roll.

the exposure desired. The skin is not included in this V-shaped incision. It has been found that this added incision in the membrane more than doubles the exposure of the larynx, making strong retraction unnecessary.

The original straight incision through the thyroid cartilage has now been transformed into a Y-shaped one, with the V part of the Y placed in the thyrohyoid membrane.

If one bears in mind the anatomy of this region and places this incision as indicated, the internal branch of the superior laryngeal nerve and the laryngeal branch of the superior thyroid artery will have been avoided. There is nothing now to interfere with the retraction or rotation outward of each half of the larynx. Instead of working through a narrow opening, one has considerable room to carefully inspect and work in the interior of the

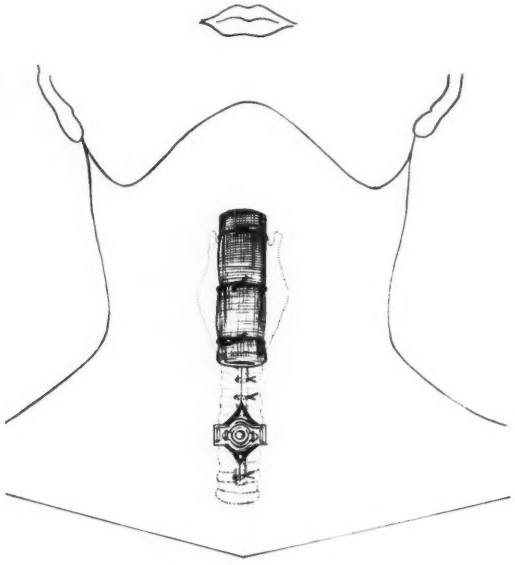

Fig. 13.—Showing gauze roll tied in place. This prevents it from slipping with neck movements.

larynx. This technic reduces the dissection of the growth with normal tissue to a simple procedure. All bleeding points after removal of the mass are easily seen and ligated.

Before dissecting the soft tissues and growth from the cartilage, a light gauze strip packing inserted into the trachea through the laryngo-fissure will prevent any secretions and blood from getting into the trachea.

REMOVAL OF GROWTH.

The dissection of the diseased tissue from the cartilage is best begun with a knife, making sure that the plane of cleavage between the perichondrium and cartilage is reached. Having properly started the dissection, a Freer separator or any thin blunt dissector is used to continue the separation. This dissection must go wide of the growth, especially in the subglottic area, for it is here that one may not take enough normal tissue beyond the growth to insure a complete removal. Then by means of curved scissors the entire mass is removed, including with it or not, as the case may require, the vocal process or more of the arytenoid cartilage.

All active bleeding can now be easily visualized and controlled by ligature or transfixtion. Oozing is controlled by pressure.

FEEDING TUBE.

All bleeding having been controlled, the introduction of the feeding tube through the nose is made surer if done now before closure of the larynx. The tube can be easily guided into the esophagus under direct vision.

CLOSURE OF WOUND.

Before closing the wound all blood and secretions are aspirated from above the tracheal packing. Then the packing is removed. Should the lower end of this packing be stained with considerable blood, then it becomes necessary to aspirate anything which may have escaped beyond the packing.

At no time has it been found necessary to suture the incision in the thyrohyoid membrane. The two halves of the thyroid cartilage are allowed to come together. The muscles and then the fascia are brought together with interrupted sutures. The skin wound is now closed by means of the so-called Stewart suture. A small gauze roll dressing is kept in place by means of stay sutures.

The upper and lower parts of the tracheotomy wound are now ready for closure (after a tracheotomy tube has been introduced), and the usual dressings applied.

POSTOPERATIVE CARE: FEEDING.

If a feeding tube has been introduced into the esophagus, the patient is fed through it. If no tube is used and the patient cannot swallow without spilling food into the larynx, then a stomach tube may be introduced through the mouth at each feeding.

It has been found, however, that while a patient may not be able to swallow fluids, he will be able to take soft things, such as jello, custard, etc. The taking of fluids is often made possible if the patient is made to suck them through a glass tube while bending forward.

TRACHEOTOMY TUBE.

This is removed as soon as possible. If the reaction is mild (as it usually is), then it can be removed in a few days. While in situ, however, careful and constant suction is to be used through the tracheotomy tube.

SUTURES.

All sutures are removed between the fifth and seventh day and the patient allowed out of bed as soon as possible after the operation.

It is worthy of note that by using the two separate incisions the thyrotomy wound heals by primary union.

While performing the preliminary tracheotomy entails an added procedure, still it is considered safer by many. Though a laryngofissure without a preliminary tracheotomy has its advocates, it is nevertheless a known fact that not infrequently these operators have found it necessary to perform an emergency tracheotomy during the operation or a day or so after.

The preliminary tracheotomy should be considered in the light of an added margin of safety, so that when its function has been fulfilled and there is no further need of a tube it may be promptly removed.

It is also worthy of mention that indirect examination of the larynx barely shows the incision placed in the thyrohyoid membrane.

CONCLUSIONS.

1. These modifications are based on a minute and careful study on forty cadavers, supplemented by actual operative experience.

2. The procedures are anatomically sound and surgically safe.

3. However, they are not intended as a panacea. Any improvement in technic should not and cannot supersede the importance of early diagnosis.

4. This operation is not recommended in advanced intrinsic cases of carcinoma.

5. Preliminary tracheotomy should be looked upon as an added margin of safety.

6. Old writers have casually stated that the surgeon could extend the thyroid cartilage incision either up into the thyrohyoid membrane or down into the cricothyroid membrane, but no definite recommendation as a routine measure was made.

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XCIII.

Clinical Notes and New Instruments.

CARCINOMA OF THE ESOPHAGUS ENGRAFTED UPON A LYE STRICTURE OF TWENTY-SIX YEARS' DURATION.*

MERVIN C. MYERSON, M. D.,

NEW YORK.

The occurrence of carcinoma of the esophagus at the site of a stenosis due to the ingestion of a corrosive is extremely rare. The esophagus immediately above a stricture is prone to chronic irritation and inflammation. This is also true of the esophagus in those portions where it is physiologically narrowed. It is also well known that carcinoma usually occurs in those areas of the esophagus which are so normally constricted. Leukoplakia is frequently seen in the region of carcinoma of the esophagus, and it is likely that the leukoplakia was a pre-existing lesion in at least some of these cases. Guisez has had patients under his care for years because of esophageal spasm and esophagitis. They have presented themselves because of a sudden increase in their symptoms and upon esophagoscopy were found to have a carcinoma.

CASE REPORT.

A man of twenty-nine was referred by his family physician with the following history. At the age of three he had accidentally swallowed lye in solution. He was gastrostomized by Dr. George E. Brewer, who also passed bougies by the retrograde method by means of a continuous string. Bougies were passed at varying intervals until the age of nine. One year ago a chicken bone lodged in the upper portion of his esophagus and was pushed down by means of a rubber tube. Recently a piece of meat would occasionally become caught and would either work its way down or it would be regurgitated. He had recently complained of a dull sensation and a sense of obstruction localized to the second thoracic level.

*Presented before the American Bronchoscopic Society, Washington, D. C., May 10, 1933.

Fig. 1. Roentgenogram showing region of stenosis in the upper esophagus due to lye ingestion 26 years ago.

Esophagoscopy revealed a moderate degree of decrease in the size of the lumen at a point 28 cm. from the upper tooth mark (Fig. 1.). The mucosa was replaced by fibrous tissue so that a firm lip presented posteriorly. Most of this lip was irregular in appearance, showing a slight elevation, but no ulcerative or granulating surface. Although there was nothing definite in the appearance to signify that a carcinoma was present, a piece of tissue was removed for study.

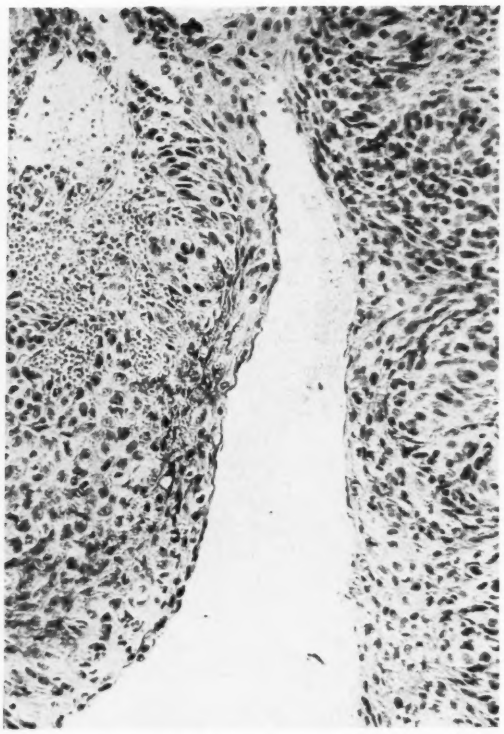

Fig. 2. Photomicrograph of tissue removed from area of stenosis illustrated in Figure 1. Diagnosis: Carcinoma.

Dr. Leila C. Knox reported as follows: "Sections from the tissue removed show very satisfactorily a portion of the muscular wall in which there is an extensive infiltration with epithelial cells derived from the squamous portion of the mucous membrane. These infiltrate in all directions in an obviously malignant fashion. There is a little differentiation, but no well marked pearls, and the tumor is closer to the squamous than to the columnar morphology. Diagnosis: Epithelioma" (Fig. 2).

The patient has been receiving deep X-ray therapy during the five months since he was esophagoscoped. There has been no change up to the present time.

136 E. 64TH ST.

XCIV.

PRESENTATION OF A CASE OF ESOPHAGEAL DIVERTICULUM.*

JOSEPH C. BECK, M. D.,

CHICAGO.

I was substituting for Dr. McGinnis at the hospital, and a male patient was referred to me with an esophageal diverticulum. The interesting point was that in looking for relief this patient had a previous operation performed externally because the surgeon thought it was a tumor. This diverticulum had a very large and very firm opening in the posterolateral part of the esophagus, right below the cricoid. The resident esophagoscopist removed a piece of tissue, and the pathologist called it a precancerous growth. At any rate, the point I wish to bring out is that it was diagnosed as a tumor from the esophagoscopic examination. Following removal of the tissue a great deal of radium was implanted in this area, and the general surgeon attempted to operate on the tumor in the neck, which he said was angiomatous because it occasionally almost disappeared. However, all he did was to explore. Then came the real diagnosis. The fluoroscopic examination was interesting to watch and see the filling of this diverticulum and the nice overflow, which was a trickle rather than a flow of material.

This man did very much better with solid food than liquid, because the liquid filled this diverticulum up so it almost obstructed the rest of the esophagus; with the solid food the filling would limit itself and some food would pass.

(Slide) The point I wish to stress in this case is, in looking over the work done (Leahey of Boston leading off, then a man by the name of Precechtel of Prague, Judd of Rochester, Minn., and others) I have not found the trick which was used in the secondary operation mentioned, namely, where one would not know just how dense the wall of the diverticulum was. Some-

*Presented before the American Bronchoscopic Society, Washington, D. C., May 10, 1933.

times they are thin and then again they are very thick. We inserted a fairly thick rubber tube into this diverticulum, and it did extend quite low down into the neck. Dissecting down to the diverticulum, the forceps grasped the tube with the wall of the diverticulum and that allowed complete dissection and the upward placing of the same, according to Leahey, very nicely. I do not know what we will do with the stiff and large opening of the diverticulum at the esophageal wall. I do not find much said in Dr. Leahey's work on this point. Strictures are likely to be so severe after doing a thorough resection of the stiff opening of the diverticulum, but my experience in laryngectomies teaches me that no matter how narrow the esophagus is the food dilates it again, providing there is not much cicatricial formation.

185 N. Wabash Ave.

XCV.

FOREIGN BODY EMBEDDED IN THE LARYNX.*

T. H. Halsted, M. D.,

Syracuse, N. Y.

This is a case of what I suspect to be a foreign body embedded in the larynx or the esophagus, and is presented for diagnosis. A woman, 43 years of age, consulted me two months ago because of aphonia. She had no pain, no secretion, no cough, no dysphagia and the aphonia was pretty nearly constant. A Wassermann was negative and chest negative. The history was that the aphonia had begun last August, about eight months ago. On examination the nose, nasopharynx and pharynx were negative. The epiglottis was more or less immovable in the vertical position. The cords could not be seen in their entirety, the middle thirds only being seen and nothing adventitious here. The right cord was not always freely movable, being at times in the position of paralysis of the right cord. This, however, was intermittent. I suspected when I first saw her a carcinoma because of the paralysis. But there was no pain to the ear, no swelling of the aryepiglottic fold and the paralysis was not a constant one.

A direct laryngoscopy under cocain left the examination unsatisfactory. On the fourth visit, finding the cord as at the first visit, I sent her to the hospital for an X-ray of the larynx and mediastinum and a barium of the esophagus. Following this she went to the operating room, and I again examined the larynx under ether and under direct laryngoscopy. This examination showed the cord fixed and a pushing of the trachea to the left side.

Roentgenograms, however, showed a fusion of the third and fourth vertebræ and what appeared to be a foreign body a quarter of an inch long by one-sixteenth in width lying horizontally at about the cricoid in the tracheo-esophageal wall. I thought

*Presented before the American Bronchoscopic Society, Washington, D. C., May 10, 1933.

the fusion of the vertebræ and the apparent foreign body must have occurred following some accident and questioned her regarding the possibility of this. She had previously said she could not recall any accident, but on the discovery of these conditions she remembered that last summer, while driving in the midst of city traffic, her husband pulled the car up suddenly to avoid a truck and struck a telegraph pole, breaking his nose and causing minor injuries to his face, which accounted for her not paying much attention to herself. She was in the back seat and was thrown forward and downward, and was a little dazed for a minute, after which she was all right. There was no evidence of a local injury and no blood was coughed up or swallowed so far as she knew.

The X-ray films which I pass around for your inspection show the fusion of the third and fourth cervical vertebræ and what appears to be a foreign body about a quarter inch long and a sixteenth of an inch in width, lying horizontally in the laryngo-esophageal wall. This latter may be a calcification of one of the laryngeal cartilages. What is the cause of the aphonia?

I confess I do not know whether this is merely a coincidence and the foreign body a myth, and the fusion of the vertebræ a congenital condition or one resulting from an early accident. The theory of the foreign body is that it was thrown off at the time of this injury, less than a year ago, and became embedded in the larynx, interfering with the movement of the cord.

405 Fayette Park.

XCVI.

CONGENITAL MALFORMATION OF THE ESOPHAGUS: REPORT OF A CASE.

CHARLES J. IMPERATORI, M. D.,

NEW YORK.

There are two forms of congenital malformation of the esophagus with several variations. In one type there is an upper and lower dilatation of the esophagus connected by a fibrous cord, and the other type is essentially the same, but instead of the cordlike connection between the upper and lower pouches, usually the lower part of the esophagus connects with the trachea. The upper pouch may also connect with the trachea. Complete absence of the esophagus is very rare.

Diagnosis is made by the objective signs—that is, these infants become cyanotic when food is taken, or in those types of malformation of the upper pouch, regurgitation takes place during or soon after feeding.

Roentgenograms with contrast mixture show the characteristic pouching, with or without the mixture in the airway. The pouch resembles a diverticulum and its lower part is usually in the region of the jugulum.

Roentgenograms showing air in the stomach would be diagnostic that there was a communication with the airway, and very likely that the lower pouch was connected with it. This has been developed by E. C. Vogt in a communication in the *American Journal of Roentgenology*, November, 1929, and also spoken of by F. T. Hill in Transactions of the American Laryngological, Rhinological and Otological Society, 1932, page 327.

Congenital tracheo-esophageal fistulæ are usually fatal (Jackson).

Haubold describes a procedure suggested by Von Hacker,[1] of exclusion of the cardia after gastrostomy. The same author describes a procedure suggested by Thomas, of tunneling between the pouches if they are sufficiently close together.

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*Presented before the American Bronchoscopic Society, Washington, D. C., May 10, 1933.

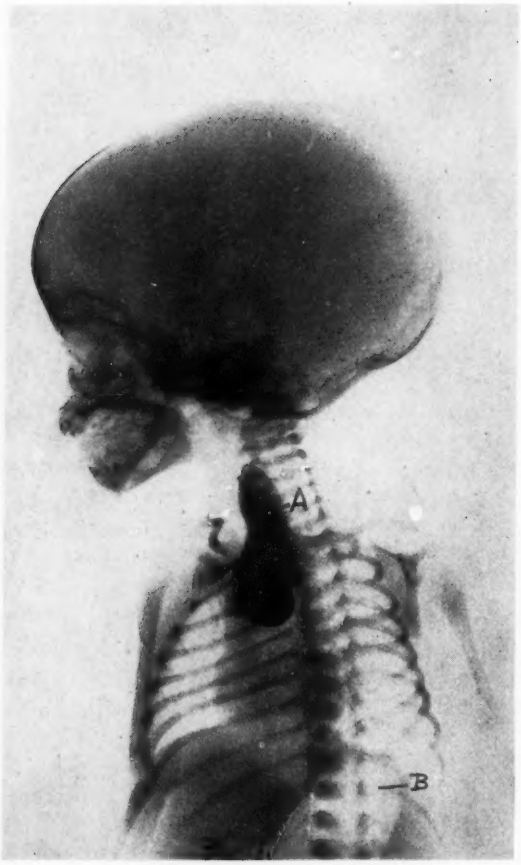

Fig. 1. Child 3 days old when this roentgenogram was taken. The barium was later removed from the pouch. *A*, Upper pouch of the esophagus. There is no communication with the airway. *B*, Stomach filled with air. This indicates that air must necessarily have reached the viscus from above.

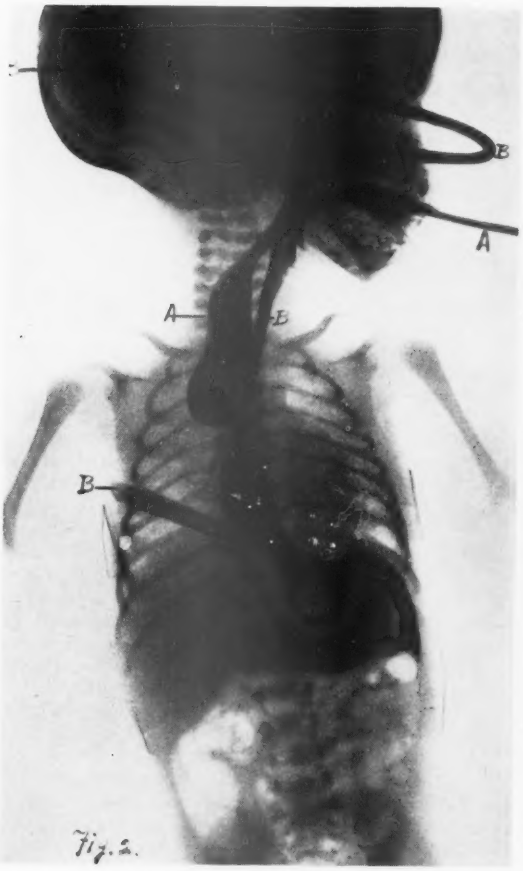

Fig. 2. This roentgenogram was made postmorten. Barium was introduced through the gastrostomy. *A*, Catheter introduced through the mouth into the upper pouch which is filled with barium. *B*, Catheter introduced through gastrostomy, into stomach, through cardia, into lower esophageal pouch, thence into the trachea, through the glottis, the nasopharynx and out of the nose.

Jackson, Mosher and others[2] have tunneled through an imperforate stricture, and it would seem feasible if carried out in the biplane fluoroscope, and using two esophagoscopes and the pouches coated with lipiodol, the elements of precision and safety would be greatly enhanced.

The patient, the first born of healthy parents, was seen when a few days old. The history given by the parents was that the child had difficulty in retaining food, and that on suckling, fits of coughing would supervene until what milk it had swallowed would be regurgitated.

A roentgenogram showed a pouching of the esophagus that did not communicate with the airway (Fig. 1-A). Air was seen in the stomach and it was assumed that there was a dilatation of the lower esophagus with a communication with the airway (Fig. 1-B).

Immediate gastrostomy was advised. Nothing further was heard regarding the patient until two days afterward when the patient was taken by the father to the Jackson Bronchoscopic Clinic in Philadelphia, where the same treatment was advised. Two days later a gastrostomy was done and it was found by the surgeon that there was a communication between the lower esophagus and nasopharynx, for a No. 8 French catheter could be passed from below, upwards. Gastrostomy wound was closed, for it was considered by those who did the operation that the esophagus was patent.

The patient was first seen at this phase of the whole procedure, advice having been given by telephone for the patient was at some distance from New York. The roentgenograms were brought to the office and a diagnosis was made on them. Dr. Jackson communicated by phone after his examination.

The patient was a very small infant, apparently perfectly formed to external appearances but slightly jaundiced and presented an appearance of dehydration. No temperature. Pulse 120. Respiration 40. Advised reinsertion of gastrostomy tube and feeding the mother's milk in sufficient amounts, Q.2.h., and external heat and immediate transfusion. Ten cc. of glucose solution had been given endermically since birth at 6 to 8 hour intervals. All these procedures were of no avail. The infant died within a few hours, having lived for 10 days.

The autopsy showed a fusion of the trachea and esophagus at third thoracic vertebra (Fig. 2). The other viscera were normal. There was no laryngeal obstruction. There was a bronchopneumonia.

In the majority of the older text books describing these conditions, the length of life is given as about 13 days.

108 East 38th St.

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XCVII.

FOUR UNUSUAL CASES.*

E. G. Gill, M. D.,
Roanoke, Va.

CASE REPORTS.

The first case, patient age 1 month, was referred to our hospital February 29, 1932, by Doctors Bishop and Hurt. The parents gave a history of the child being restless, crying and refusing to nurse for the past three days. At the end of the second day there was distinct wheeze and a slight embarrassment of the respiration. The family physician felt that an enlarged thymus gland was causing the symptoms and suggested an X-ray study. The X-ray plates showed a safety pin in upper esophagus, point up (Fig. 1). Operation: With the use of the infant size direct laryngoscope, the pin was removed. The point of the pin was embedded in the under surface of the left arytenoid cartilage. This was responsible for the laryngeal symptoms. All symptoms subsided immediately following the removal of the pin. The parents have no idea how the accident occurred nor was foreign body aspiration suspected by the parents or the family physician. This case demonstrates the importance of having X-ray plates made in every case where there is difficulty in swallowing or breathing regardless of the patient's age. An infant one month of age does not have co-ordinated use of his hands, consequently the accidental swallowing of the pin is of unusual interest.

Case 2, patient age 22 months, was admitted to our hospital on September 24, 1932. The parents gave the following history: Thirty-six hours prior to admission to the hospital the child was eating squirrel meat. The child choked, vomited and turned blue. The child had not swallowed anything, even liquids since the accident. Twelve hours following the accident the child's breathing became difficult. On admission the child was in an extremely cyanotic condition and dyspnea was marked. With direct laryngoscope the larynx revealed a marked edema of the arytenoids. A 3½ mm. bronchoscope was introduced and the bronchi were found to be clear. Realizing that a tracheotomy was indicated, this operation was done while the bronchoscope was in the bronchus. The cyanosis and dyspnea was immediately relieved. Six hours later an esophageal speculum was introduced and at the level of the cricopharyngeus the vertebrae (of a squirrel) was seen and removed. The removal was difficult due to the swelling of the mucous membranes. Feeding by a lavage tube was necessary for four days following the removal of the foreign body from the esophagus. The swelling of the mucous membrane was so extensive as to prevent the swallowing of liquids. The point of interest in this case is that a large sharp pointed object in the esophagus causes rapid swelling of the tissues in twelve hours sufficient

*Presented before the American Bronchoscopic Society, Washington, D. C., May 10, 1933.

to produce marked embarrassment of respiration and the inability to swallow even liquids. Tracheotomy tube was worn nine days (Fig. 2).

Case 3, patient age 29. We received a telephone call an hour before admission from a physician stating that he was sending in an emergency foreign body case. Fluoroscopic examination revealed a large shoe horn in the patient's larynx. From this description we felt the foreign body must be in the esophagus, as it was too large to enter the larynx. The patient on admission was in an extreme dyspnea and in a state of shock. With the use of the direct laryngoscope, the end of the shoe horn could be seen within the larynx. The object was firmly impacted and required considerable effort to remove it. Due to the traumatism of the larynx, we felt that a tracheotomy in a few hours would be necessary. The patient's breathing was slightly labored for twenty-four hours following the operation. Hoarseness was marked for one week, otherwise the recovery was uneventful. This patient had attempted suicide several times and had been under the care of a constant attendant for a long period of time. While in his room alone for only a few minutes he attempted suicide with the only weapon he could find. He stood in front of a mirror and lifted up his epiglottis with one hand and forced the shoe horn into his larynx with his other hand. This patient was a graduate of one of the leading universities of this country and was a very intelligent man. He realized that swallowing the shoe horn would not accomplish his purpose and he deliberately placed it into his larynx (Fig. 3). We are extremely sorry we do not have an X-ray plate to demonstrate the location of this object, and our reason for not making one was due to the extreme dyspneic condition the patient was in when admitted to the hospital. We feel that the case is of interest in that one can introduce so large an object into his larynx without causing serious damage. This patient a few weeks later committed suicide by cutting his throat with a pair of scissors.

Case 4, patient age 15. Patient gave a history of having swallowed a screw head from a banjo three years ago. Immediately following the accident the patient had the usual symptoms of a foreign body aspiration, choking and coughing. These symptoms were later followed by other chest symptoms which finally led to the diagnosis of pulmonary tuberculosis. During this period of time he was not X-rayed. After he was referred to the Catawba Sanatorium about one month prior to admission to our hospital, the X-ray plates were made and revealed the presence of a foreign body in the left inferior bronchus (Fig. 4). This is another of the many cases of obscure chest symptoms that could be easily diagnosed if given the benefit of an X-ray examination. This case presented a number of difficulties as it had migrated to the distal portion of the bronchus and required a bronchoscope 5 mm. by 45 cm. to reach it. The usual size bronchoscope for a patient this age was entirely too large to enter the small bronchus. The object was firmly impacted with granulation tissue around it. This is a case where a biplane fluoroscope would have unquestionably been of aid and would have shortened the time of operation. The chest symptoms have all subsided since the removal of the foreign body.

711 South Jefferson St.

XCVIII.

TRACHEO-ESOPHAGEAL FISTULA, PROBABLY CONGENITAL, IN A WOMAN 58 YEARS OF AGE.*

RICHMOND MCKINNEY, M. D.,

MEMPHIS.

CASE REPORT.

Mrs. H. L. McD., aged 58, was first seen October, 1930. This patient had had a general physical examination, and gave a history of having for the past twenty-five years been suffering with sick stomach and vomiting spells, and recently had been troubled with paroxysms of coughing. There was some loss of weight, but no fever and no pain of any kind. The cough was described as being much worse after meals, when, to use her language, she gags and spits up food. Her diet was confined mainly to liquids and semi-liquids. She could not rest on her back, resting better on her left side. Sputum examination revealed no tubercle bacilli in the thin creamy sputum which was obtained. The sputum was negative for tubercle bacilli with the concentration (antiformin) method. Wassermann was negative. It was thought that since she was undernourished, nervous and apprehensive, she did not get enough food on account of her fear of coughing.

Chest films were negative, but on account of the marked distension of the abdomen, a gastrointestinal study was undertaken. X-ray pictures were then made, and barium was noted in the right bronchial tree.

Mrs. McD. was sent to me for diagnostic bronchoscopy and esophagoscopy, she bringing with her a very excellent set of films of the chest, made at intervals. On bronchoscopy, under local anesthesia, I found at the lower end of the trachea, on the right side, a small amount of creamy fluid apparently coming through the tracheal wall. This was wiped away, and slowly would reform. The suspected opening apparently was very small, as it could not be determined accurately by means of the bronchoscope. Subsequent ingestion of barium milk gave the same roentgenologic picture as that sent with the patient. Esophagoscopy was, as would be expected, negative, owing perhaps to the rugae of the walls, making it difficult to determine the presence of an opening so small.

So far as I could ascertain, and admitting the possibility of error, it seemed that there was a minute tracheo-esophageal fistula, too small for large food particles to invade the trachea and thence enter the lung, and coming through in such small quantities that the lung had developed a resistance to this foreign material, which had prevented the development

*Read before the Southern Section Meeting of the American Laryngological, Rhinological and Otological Society, February 13, 1933, Memphis, Tenn.

of a pneumonia or lung abscess. Furthermore, the history of at least twenty-five years of sick stomach and vomiting, and latterly paroxysms of coughing, at least justified a reasonable suspicion that this was of congenital origin, although, of course, there are no grounds for a positive opinion in this connection.

Since this case came to my attention I have been scanning the literature to see if I could learn of authentic cases of tracheo-esophageal fistula without the prompt death that we expect in such cases. There are, of course, numerous cases of tracheo-esophageal fistula reported in the newly born, but these, so far as I can learn, have promptly gone to exitus. My search has not been fruitful of a great deal of success, doubtless due to the fact that I am not closely in touch with extensive sources of reference, but in the Journal of Laryngology and Otology, for March, 1929, Mr. V. E. Negus of London reports a case of congenital opening into the trachea in a man 45 years of age, who died of general weakness due to a carcinoma, which was supposed to be primarily in the esophagus.

On postmortem examination a congenital opening was found, but it had not produced any symptoms in life, and had not given rise to pneumonia. The opening led in a downward direction from the trachea to the esophagus, and on the esophageal aspect there was a small, valvelike hood of mucous membrane, which must have prevented the regurgitation of food into the trachea.

In the Transactions of the American Laryngological, Rhinological and Otological Society for 1916, Dr. Robert Levy has a paper entitled "Esophagotracheal Fistula," in which there is quite an extensive review of the literature of this subject. Most of the cases reported, of which there were quite a number, went to an early death, but many had incomplete histories, so the results were not known. A majority of the cases reported were congenital, but some of them were acquired.

In a personal communication from Dr. Chevalier Jackson, in which he cites, with his customary thoroughness, a list of references in which the subject of tracheo-esophageal fistula is considered, several of which are not now available to me, he says: "In babies the condition is not exceedingly rare. In adults, however, it is almost unknown as a proved fact. In a number of the

cases that I have seen in adults and older children we could not be certain of the etiology; we suspected in some of the adult patients that the basis of the condition was congenital and that a thin, membranous layer of tissue had occluded it enough to prevent entrance of swallowed food and liquids into the trachea until adult life; but we were never able to prove this."

In my case I am frank to state that the only way in which a positive diagnosis could be made doubtless would be through an autopsy, which event I trust may long be deferred for this good woman's sake, although if she were available to me for regular study it might be that careful bronchoscopy, after the ingestion of, say, one of the anilin dyes, might confirm or negate my description.

At any rate, this case is most interesting, and I hope to make further reports on it at a later date.

899 MADISON AVENUE.

XCIX.

SUBCUTANEOUS EMPHYSEMA FOLLOWING TONSILLECTOMY.

J. Chas. Dickson, M. D.,

Houston, Texas.

Subcutaneous emphysema following tonsillectomy is extremely rare, if the number of cases reported can be taken as indicative.

F. M., female, aged 17, entered Herman Hospital with a history of recurring attacks of tonsillitis. She also had a condition diagnosed by the dermatologic service as angioneurotic edema. Examination was negative except for a pair of chronically inflamed tonsils and a dermographia. Tonsillectomy and adenoidectomy was done. She took the anesthetic fairly easily and there was nothing unusual about the operation. The bleeding was easily controlled and she was sent back to the room in good condition. Forty minutes later there occurred marked subcutaneous emphysema, extending from the lower margin of the ribs, front and back, to above the ears. We were unable to find any pathology other than the emphysema. X-ray of the chest showed heavy bronchial markings, indicating chronic inflammation.

The recovery was uneventful, the emphysema disappearing in five or six days. The patient's only complaint was soreness over the chest.

Examination of literature showed very few cases of this nature reported. (See bibliography.)

All the operations were performed under a general anesthetic. The emphysema was noticed some time within an hour after the operation. All recovered with no special treatment.

There were a number of theories as to the mechanism of production. Van Hofe,[7] Governale,[1] Richards[3] and Rosenheim[5] believe that the alveolus ruptures, the air passes to the hilus of the lung along the bronchioles and bronchi, then along the large vessels to the neck and from there into the subcutaneous tissue.

Von Hofe,[7] Stein,[6] Parish[2] and Richards[3] believe that it passes directly through the tonsil fossa bed to the subcutaneous tissue.

There is little difficulty in diagnosis.

The treatment is to do nothing.

622 Medical Arts Bldg.

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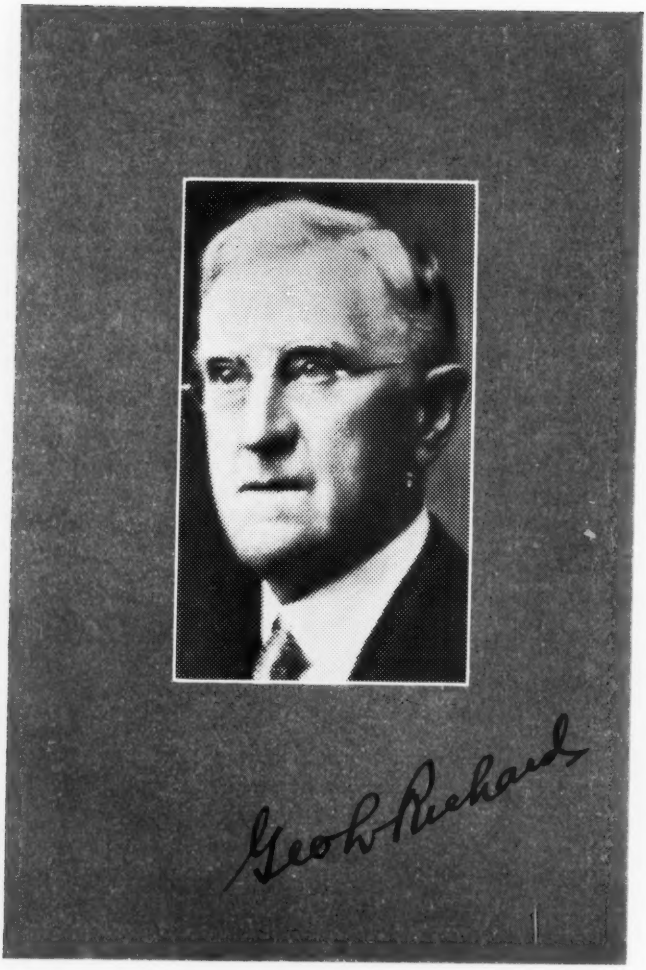
NOTICE.

THE AMERICAN BOARD OF OTOLARYNGOLOGY.

An examination was held in Boston, Mass., September 16, 1933, during the meeting of the American Academy of Ophthalmology and Otolaryngology. Seventy-nine candidates were examined, of which thirteen were conditioned or failed.

The Board will hold an examination in Cleveland, Ohio, June 11, 1934, during the meeting of the American Medical Association. Also, early in the summer (date is not set) an examination will be held in Butte, Mont., in connection with the meeting of the Pacific Coast Oto-Ophthalmological Society. Prospective applicants for certificate should address the Secretary, Dr. W. P. Wherry, 1500 Medical Arts Building, Omaha, Neb., for proper application blanks.


Geo W Pickard

GEORGE LYMAN RICHARDS.

Dr. George Lyman Richards was born in Unionville, Connecticut, on June 1, 1863, the son of a Connecticut farmer and miller. His early boyhood days were spent on a farm and much of his spare time was spent as a helper in the grist mill of his father. He received his early education in the local grammar and high schools, following which he spent a single year at Williston Seminary at East Hampton. There then followed a year at the business college of Bryant and Stratton in Boston, a period of time which he always regarded as invaluable, even in the profession of Medicine.

In the fall of 1883 he entered the Harvard Medical School, where for three years he pursued the study of medicine under such illustrious teachers as Thomas Brice, J. C. Warren, the elder Dr. Fitz and the elder Dr. Cheever. Following his graduation from the school in 1886 he took an interneship at the Rhode Island Hospital in Providence. Following this he then began his work as a country practitioner in the town of East Greenwich, R. I., taking the place of the local doctor who was absent on a trip to Europe. His next move was to the town of Marion, Mass., where he practiced general medicine for several years up until 1893 when, anxious to confine his work to a more particular field, he sailed with his sister for Europe and matriculated as a student in the University of Holland. Here he laid the foundations for his work in the specialty of otolaryngology, which he pursued up to almost the time of his death.

Moving to Fall River, Mass., in 1896, he engaged in the active practice of his specialty and in addition maintained offices in the adjacent cities of New Bedford and Taunton, during which period he conducted a most active practice between these various cities. He early affiliated himself with numerous medical societies, both general and special, joining the Chirological Society in 1898 and serving as its president in 1918. He was also a member and one-time president of the New England Otological and Laryngological Society, a member of the American Otological Society, the American Laryngological, Rhinological and Otological Society

and the American Medical Association. He was also a life member of the American College of Surgeons. He was keenly interested in local medical affairs and for many years served as librarian of the local medical society, in Fall River.

Dr. Richards was an ardent traveler and made numerous trips to Europe and on two occasions made the circuit of the entire globe. On one occasion he was a delegate to the British Congress on Tuberculosis, and in 1919 he went as a member of the Near East Relief Expedition to European and Asiatic Turkey. He was subsequently made medical director of this expedition and in this capacity visited all of the relief stations throughout the greater part of Asiatic Turkey, making a report on the work being done by this organization of great interest and importance. The two trips around the world included visits to many of the most important foreign missionary stations maintained by the American Board for Commissioners of Foreign Missions, of which he subsequently became one of the members of the prudential committee. Visits to these far-away mission stations took him to some of the remote portions of India, China and Japan, where he conducted clinics and performed many operations, teaching students and giving courses in his special line of work. On one occasion he performed a mastoid operation 3000 miles up the Yangtze River. Missionaries returning to this country have frequently reported the great benefit derived from the work which Dr. Richards did at their native stations and the impression made upon the local government by the interest which it aroused.

During the war he sat on the Board of the Examination of Applicants for Exemption from the Draft. His early religious training at home found expression in his after-life when he was at one time both treasurer and deacon in his own Congregational Church.

Dr. Richards' interests, other than those in the field of medicine, covered a wide range of activities. He was an ardent gardener, a great lover of tools and mechanical appliances, being ever on the lookout for some new article along these lines. He was an omnivorous reader, particularly on books of travel, and possessed a library which covered an amazingly wide range of literature. He was himself the author of a book entitled "Nose and Throat

for the General Practitioner," published by Paul Hoeber of New York. His greatest literary efforts, however, were applied to the annual production of the Transactions of the American Laryngological, Rhinological and Otological Society, of which he was the editor for many years. This arduous task of yearly production of an ever increasing sized book he carried out with the greatest of pleasure and with enormous pride in the result.

His capacity for intimate friendship was almost limitless, and he leaves behind him, scattered throughout the world, in all corners of the globe, those who will remember him for his personal service and stimulating friendship.

He passed away on November 9, 1933, at West Dennis on Cape Cod, where he had maintained a residence during the last three years and where he continued his medical interests at the Cape Cod Hospital in Hyannis.

PUBLICATIONS OF GEORGE LYMAN RICHARDS.

1. Consumption: Cases that Should go to Colorado and Cases that Should Stay at Home. Read before the Fall River Medical Society, October 12, 1898.

2. Perichondritis of the Larynx: With a Report of Two Cases and the Description of a New Tracheotomy Tube. Read by title before the American Laryngological, Rhinological and Otological Society, at its annual meeting in Pittsburg, May 11 and 12, 1898.

3. The Serum Treatment of Pulmonary Tuberculosis: With a Report of Two Cases Treated with Antitubercle Serum. Read before the Fall River Medical Society.

4. The Local Medical Society: A Plea for its Better Support. From the Medical News, December 31, 1898.

5. The Technique of Tonsillotomy. From the Charlotte Medical Journal, Charlotte, N. C., Oct., 1898.

6. Bleeding Polyp of the Septum (Telangiectoma); Report of a Case. From The Laryngoscope, St. Louis, December, 1898.

7. Paramonochlorophenol: Its Use in the Local Treatment of Laryngeal Tuberculosis. Presented to the Section on Laryngology and Otology at the Forty-ninth Annual Meeting of the American Medical Association, held at Denver, Colo., June 7-10, 1898.

8. The Use of Gelato-Glycerine Bougies in the Treatment of Earache. From The Laryngoscope, St. Louis, August, 1899.

9. Earache: Causes, Treatment, Relation of the Exanthemata Thereto. From the Boston Medical and Surgical Journal of July 28, 1898.

10. The Minor Surgery of the Nose and Throat. From The International Journal of Surgery, Oct., 1899.

11. The Facial Nerve in Its Relations to the Aurist. From ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, May, 1899.

12. An Unusual Case of Traumatic Rupture of the Membrana Tympani. Read before the Sixth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Philadelphia, May 31 to June 2, 1900.

13. A Brief Report of a Case of Cerebral Abscess of Otitic Origin; Operation; Death. Presented at the Annual Meeting of the American Laryngological, Rhinological and Otological Society at Philadelphia, May 31-June 2, 1900.

14. Syphilis of the Upper Air-Tract: Some Remarks on Its Diagnosis and Treatment. Presented to the Section on Laryngology and Otology, at the Fifty-first Annual Meeting of the American Medical Association, held at Atlantic City, N. J., June 5-8, 1900.

15. Is It the Duty of the State to Care for the Tuberculous Individual? The Experience of Massachusetts. From The St. Paul Medical Journal, February, 1901.

16. The Treatment of Suppurative Otitis Media in Young Children. From The Medical News, May 19, 1900. Presented to the American Laryngological, Rhinological and Otological Society (Eastern Section), at its Annual Meeting in Philadelphia, February 17, 1900.

17. The Use of Gelato-Gylcerin Bougies in the Treatment of Acute Earache in Young Children. Read at the Fifty-third Annual Meeting of the American Medical Association in the Section on Diseases of Children, and approved for publication by the Executive Committee: Drs. Edwin Rosenthal, H. M. McClanahan and Samuel W. Kelley.

18. The Relation of the Dentist to the Nose, Throat and Ear Specialist. From The International Dental Journal, Feb., 1905.

19. Empyema of the Frontal Sinus: Some Observations on Its Treatment. Presented before the Annual Meeting of the American Laryngological, Rhinological and Otological Society at New York City, 1901.

20. The Evil of Eponyms. Chairman's address before the section on Laryngology and Otology, at the Fifty-fourth Annual Session of the American Medical Association at New Orleans, May 5-8, 1903.

21. Aural Neuralgia of Dental Origin. Read before the Eastern Section of the American Laryng., Rhinolog. and Otolog. Society, Philadelphia, Feb. 4, 1905.

22. The Significance of Pus in the Nose, with Special Reference to the Affections of the Accessory Sinuses and Their Proper Treatment. Presented before the Buffalo Academy of Medicine, March 28, 1904.

23. Chronic Suppurative Otitis Media. When should radical surgery be employed in its treatment, and of what should this consist. Read before the New York Academy of Medicine, Section on Otology, April 10, 1902, also before the American Laryngological, Rhinological and Otological Society at its annual meeting in Washington, June, 1902.

24. Reflex Cough. Read before the Eastern Section of the Am. Laryngolog., Rhinolog. and Otolog. Soc. at its annual meeting, held in Washington, D. C., Jan. 28, 1899.

25. On the Relative Frequency of the Dental and Nasal Origin of Antral Empyemata. Portion of address delivered before Harvard Odontological Soc., Dec. 15, 1904, on the Relation of Dentist to the Ear, Nose and Throat Specialist and extracted from the International Dental Journal.

26. Technic of the Operation for Removal of Middle Turbinate. Presented before the Section on Laryngology and Otology of the Am. Med. Association, Atlantic City Session, June 4-7, 1907.

27. Nose and Throat Work, for the General Practitioner. International Journal of Surgery Co., New York.

28. The Non-Operative Treatment of Chronic Otitis Media Purulenta, with Special Reference to the Use of Pyoktanin. From the Laryngoscope, St. Louis, Sept., 1905.

29. The Treatment of Empyemata of the Maxillary Sinus Through the Nose. Read in the Section on Laryngology and Otology of the Am. Med. Association at the 56th annual session, July, 1905.

30. Personal Experiences with Empyemata of the Frontal Sinus. Presented as candidate's thesis, Am. Laryngolog. Association, Atlantic City meeting, 1905.

31. Atrophic Rhinitis: A Reproach to Rhinology. Read in the Section on Laryngology and Otology of the Am. Med. Association at the 57th annual session, June, 1906.

32. What Should Be the Attitude of Public Sanatoria Toward Cases of Tubercular Laryngitis; with Suggestions as to the General Plan of Treatment of Such Cases in Sanatoria. Boston Med. and Surg. Journal, Aug. 9, 1906.

33. A Review of the Methods in Use for the Removal of Adenoids, with the Description of a Method Which is Thorough, Rapid and Safe. Read before the 29th Annual Congress of the American Laryngological Association, Washington, D. C., May 7-9, 1907.

34. Coup D'Oeil Sur Les Methodes Employees pour L'Ablation des Adenoides: Description d'une Methode Radicale, Rapide et Sure. Arch. Internationales de Laryngologie.

35. Circumscribed Purulent Inflammation of the Labyrinth. Paper read before the German Otological Society in Bremen, May 17, 1907.

36. The Treatment of Hay Fever. From the Transactions of the American Laryngological, Rhinological and Otological Society, 1909.

37. The Present Status of the Tonsil Operation: A Collective Investigation. From the Transactions of the Am. Laryngo., Rhino. and Otolog. Society, 1909. Presented before the Am. Laryngo., Rhino. and Otolog. Society at its 15th annual meeting, Atlantic City, June 3, 1909.

38. The Relation of the Dentist to the Nose, Throat and Ear Specialist. From The International Dental Journal. An address delivered before the Harvard Odontological Society, Dec. 15, 1904.

39. Erysipelas as a Complication of Mastoid Disease. Read before the 16th Annual Meeting of the Am. Laryngolog., Rhinolog. and Otolog. Society, Washington, D. C., April 29, 1910.

40. On the Possible Effect Upon the Auditory Labyrinth of the Ehrlich-Hata Remedy in the Treatment of Syphilis. A paper presented before the Gesellschaft für Aerzte, Vienna, Dec. 6, 1910.

41. The So-called Conservative Radical Mastoid Operation with a Description of the Technique of Heath, Bondy and Siebenmann. Read before the Am. Laryngol., Rhinolog. and Otolog. Soc., Atlantic City, June 2, 1911.

42. Acute Otitis Media. Read before the Eastern Med. Soc. in a Symposium on Practical Features of Ear Disease, at the N. Y. Academy of Medicine, Sept. 20, 1911.

43. A Review of 81 Cases of Inflammation of the Frontal Sinus: A Comparative Study. Read before the annual meeting of the Am. Laryngological Association, Philadelphia, May 30, 1911.

44. Acute Otitis Media—How Best to Treat It so as to Prevent Complications. Read before the Eastern Med. Soc., Sept. 20, 1911; N. Y. Med. Jour., Oct.. 1911.

45. Report of a Case of Phlegmon Starting as a Peritonsillar Abscess and Extending Downward as Far as the Second Ring of the Trachea. Read at the 35th Annual Meeting of the Am. Laryngological Assoc., Washington, D. C., May 6, 1913.

46. Peritonsillar Abscess Extending Downwards as Far as the Second Ring of the Trachea. From the Boston Med. and Surg. Journal of Aug. 21, 1913.

47. Technic of Tonsillectomy. Read in the Section on Laryngology, Otology and Rhinology of the Am. Med. Association at the 64th Annual Session, held in Minneapolis, June, 1913.

48. Relations of the Tonsils, Adenoids and Other Throat Conditions to Tuberculous Cervical Adenitis. Delivered before the Massachusetts Med. Society, June 10, 1914.

49. Diseases of the Ear, Nose and Throat Observed in Asia Minor. From The American Journal of Surgery, May, 1920.

50. Foreign Body in the Lung: Report of a Case in Which the Primary Diagnosis was Suggested by a Blood Examination. Presented before the 37th Annual Meeting of the Am. Laryngological Association, Niagara Falls, June 3, 1915.

51. A Review of Recent Progress in Otology. Read before the 21st Annual Meeting of the American Laryngological, Rhinological and Otological Society in Chicago, June 15, 16, 17, 1915.

52. Report of a Case of Brain Abscess with Unusual Findings. Read before the American Otological Society at its 48th Annual Meeting at Niagara Falls, June 4, 1915.

Abstracts of Current Articles.

NOSE.

Anatomic, Pathologic and Histologic Investigations on the Ethmoidal Nasal Mucosa in the Anaphylactic Bronchial Asthmatic (Indagini anatomo-pathologiche ed isto-funzionale sulla mucosa nasale ethmoidale degli asmatici bronchiali anafilattici).

Rubaltelli, E. (Padova), Arch. Ital. di Otol., Rino. e Laryng., 44:77 (February), 1933.

The author has conducted histopathologic and chemicodynamic investigation on the ethmoidal nasal mucosa of patients suffering with anaphylactic bronchial asthma; in these patients the mucosa presents, according to his previous article published in this same journal (43:73, December, 1932), the typical characteristic changes well illustrated by a colored picture accompanying this article. He reports in detail some of his experiments and final results; the three microphotographs demonstrate the histologic changes. He finally concludes as follows: The histopathologic investigations show:

1. In the Ethmoids of Periodic Asthmatics Examined During the Period of Exacerbation.—Epithelial hyperplasia and hypersecretion, thickening of the basal membrane, slight hypertrophy of the glands, albuminous exudation, eosinophiles, turgescence of the blood vessels.

2. In the Ethmoids of Periodic Asthmatics Examined During the Period of Quiescence.—Epithelial metaplasia, thickened basement membrane, proliferation of supporting cells, slight glandular atrophy, deep localized exudative edema.

3. In the Ethmoids of Chronic Perennial Asthmatics.—Edematous infiltration of the deep interstitial tissue and a tendency of the submucosal tissue to acquire a reticular construction; therefore sclerosis and gradual atrophy. Constant thickening and hyaline changes in the walls of the blood vessels.

The chemicodynamic investigations, which were performed by applying to the nasal mucosa adrenalin, cocain, procain, optochin, atropin, pilocarpin and amyl nitrate, have demonstrated that the characteristic shiny-white color of the ethmoid mucosa is prac-

tically always permanent and rarely changed by the applications of the vasoconstrictors or vasodilators. This demonstrates that the discoloration is not due to spasm of blood vessels nor to vasomotor phenomena; but is due to permanent histopathologic changes in the mucosa itself, in the capillary and pericapillary tissues and a lack of appropriate response, to certain stimuli, in the blood vessels' tissue proper. SCIARRETTA.

Research on Transmissibility of Ozena Between Human Beings (Récherches sur la transmissibilite de l'homme à l'homme).

Drs. A. Costinin and St. Popian (Bucharest), Otorhinolar. Internat., 17:321 (May), 1933.

Citing their failure (reported at the Madrid Congress) during two years to cause ozenal changes in animals by inoculation of ozenal material, the authors attempted inoculation of twelve persons. There was no result with simple rubbing of ozenal crusts on the turbinates, or from packing the nose with cotton soaked in ozenal material. Submucous inoculation in the inferior turbinate of 1 cc. of an emulsion of the germs found in twenty-four hour cultures, provoked a severe febrile reaction, with much local pain and swelling for several days; after evacuation of pus formed, complete recovery without ozenal changes was noted.

The formula used was ⅔ Perez, ⅓ of a mixture of Löwenberg-Abel, Hoffman, Friedländer and mucosus capsulatus.

They maintain that ozena is not microbic or contagious, and agree with authors calling it a neurovascular disease of the mucosa. FENTON.

Experimental Meningitis from the Frontal Sinus (Experimentelle untersuchungen über die vom sinus frontalis ausgehende rhinogure meningitis).

Yamamoto, Dr. S. (Fuknoka), Jap. Journ. Med. Sci. Otolar., 2:15 (June), 1933.

Working under Professor Kubo, the author brought about meningitis in cats by plugging the nasofrontal duct with paraffined cotton, then inoculating the cavity with hemolytic streptococci, also with mustard oil and tincture of iodin. When normal drainage was restored, localized leptomeningitis healed rapidly. In the presence of severe destruction of the epithelium, two paths of infection were found—one directly through the frontal.

In meningitis of marked degree several distinct pathways could be found.

This is the first time that experimental purulent meningitis has been produced from the sinus route. Apparently closure of the nasofrontal duct is the necessary factor. Changes in the membrane parallel those described two years ago by Fenton and Larsell. FENTON.

The Routes of the Respiratory Reflexes Determined by Odoriferous Substance—Importance of the Olfactory Nerve (Le vie dei riflessi respiratori alle sostanze odorose: importanza del nervo olfattorio).

Ciurlo, L. (Genoa). Arch. Ital. di Otol., Rino. e Laryng., 44:109 (February), 1933.

The author has made experiments on rabbits in which he removed the olfactory bulbs, stimulating the reflex with ammonia, sulphuric carbonate and ether. He concludes that the respiratory reflex is due to the olfactory nerve, while the trigeminal is the regulator of this reflex. SCIARRETTA.

Discussion on Acute and Chronic Inflammatory Disorders of the Ethmoidal and Sphenoidal Sinuses.

F. Lemaitre and J. Aulin. Proceedings of the Royal Society of Medicine, Vol. XXVI., No. 8, page 969.

A classification of acute and chronic inflammatory disorders of the posterior sinuses with a discussion on their etiology and treatment is given. Etiology is divided into well-known facts and debatable points. The former group includes all infections of the upper air passages, influenza, adenoids in children and fevers, especially measles and scarlet fever. Numerous causes which aid in an acute infection becoming chronic are reviewed and include swelling of the ethmoid mucous membrane, high deviation of the septum, chronic purulent infection of the adenoids and clumsy nasal douching in cases of ozena.

In the discussion of debatable points it is pointed out that while the etiology of acute and chronic purulent sinusitis may be simple enough, the etiology of nonpurulent sinusitis, especially of polypoid sinusitis and hyperplastic sinusitis, is not simple, and different theories regarding the disease are reviewed. The writers believe that the general condition of the patient plays an important part in the appearance and evolution of polypoid sinusitis. Then if an inflammatory or simply irritative process is prolonged,

the mucous membrane, which at first only reacts superficially in the epithelial layer, begins to react in the deeper layer, especially in the periosteum, which in the ethmoid particularly is inseparable from the nasal mucous membrane. The participation of the periosteum in the inflammatory process explains the lesions in the bone, the partial absorption of the bony layers, which secondarily take on the appearance of "necrosing ethmoiditis." Polypoid sinusitis and hyperplastic sinusitis are histologically an identical inflammatory process in different stages. The authors believe that the general condition is indirectly the origin of bilateral nonsuppurative ethmoiditis and is perhaps itself dependent on the action of the endocrine glands in consequence of a disorder, doubtless hereditary, such as inherited syphilis.

Regarding the medical treatment of sinusitis, a small quantity of potassium iodid liquifies the nasal secretion and helps its expulsion. In severe infections or complications vaccine therapy should not be neglected. Catheterization and lavage of the sphenoid sinus, which is often effective, should be widely employed.

The transethmoid endonasal operation accomplishes the complete destruction of the ethmoid and sphenoid focus of infection, and although unfortunately somewhat mutilating is satisfactory because it prevents any postoperative exacerbation. The endoseptal route is satisfactory in bilateral sphenoid sinusitis but is not applicable to an associated ethmoiditis. The external operation does not give results much superior to the endonasal operation and should not be employed unless there is a complication threatening.

DISCUSSION.

Mr. A. Lowndes Yates drew attention to the fact that in the posterior sinuses the results of inflammation may be far more serious than in the anterior group, and chronicity is liable to be established by reason of the lack of symptoms in cases of open sinusitis, and because the ostia of the sinuses discharge their contents on to an area of the mucous membrane which is neither in the inspiratory nor expiratory airway, and hence the sinus is not cleansed by sneezing. He describes his technic of measuring the lysozymic factor from the discharge which assists in determining the form of treatment.

Mr. Herbert Tilley agreed with the writers that chronic inflammation of the sphenoid and posterior ethmoid sinuses was much more common than was formerly believed. One of the most characteristic signs of chronic suppurative inflammation of the posterior nasal sinuses is a purulent discharge with edema of the mucous membrane in the anterior aspect of the olfactory sulcus. Nasal endoscopy and the displacement method of Proetz will often provide valuable information.

Mr. P. Watson-Williams discussed the importance, in cases of retrobulbar neuritis, of making a correct differential diagnosis of its cause. Observations have proved the sinuses sterile in cases of retrobulbar neuritis due to disseminated sclerosis, but, on the other hand, many examples of retrobulbar neuritis due to sphenoid sinusitis, which rapidly yielded to disinfection and drainage, have been demonstrated.

Mr. F. H. Whittington stated that it is not in the cases with obvious purulent infection that the optic nerve is affected, but in chronic cases in which the infection passes through the bone—often of paper-like thinness. Plastic inflammation occurs between the dura and the pia arachnoid and by the combined effect of local pressure and local toxins function is diminished. He described the signs that would lead him as an ophthalmologist to suspect some such process and therefore urge an operation.

Mr. F. C. Graves referred to an investigation into sinusitis in cases of mental disorder in the Birmingham Mental Hospitals during the last seven years and published in the Journal of Mental Science, 1932, LXXVIII, 459. Nasal sinus disease, and particularly sphenoid disease, may cause cerebral and endocrine dysfunction.

Mr. F. A. Peckworth has made a special study of the sphenoid sinus and pituitary gland in cases of mental disorder. In 317 consecutive postmortem examinations there was some evidence of chronic infection of this sinus in 100 cases. Illustrations and specimens were shown to demonstrate the possibility of chronic extension of sepsis from the sinus into the pituitary gland, the blood supply of the brain and the intercranial nerve structures.

Four illustrative cases of ethmoid and sphenoid sinusitis were presented. Goldsmith.

Progressive Lethal Granulomatous Ulceration of the Nose.

J. P. Stewart (Edinburgh), Journal of Laryngology and Otology, 48:657 (October), 1933.

In a very scholarly and thorough manner the author reviews a series of cases presenting a very unusual lesion. Unfortunately, as the title would indicate, nothing definitely is known about the disease. It affords some comfort, however, to realize this fact—even the pathologists who have examined the tissue in the different cases have not been able to reach any conclusions and rarely have been able to agree upon a common finding.

Stewart very wisely goes into considerable detail on the differential diagnosis of the lesion. Syphilis, of course, is usually the first diagnosis and also the most difficult one to release. Then comes the diagnosis of malignancy, then tuberculosis and, finally, the more unusual and rarely seen conditions such as Leishmaniasis, gangosa, agranulocytosis, rodent ulcer and granuloma venereum. There is no response to treatment, the duration of the illness is from one to two years and about all that one sees is the horrible picture of the gradual and progressive destruction of the nose, face and the pharynx, including bone and cartilage. There is usually a mild leukocytosis with the white blood cells of normal proportion and a continuous fever, and probably one of the most unsightly lesions that man could fall heir to.

In Stewart's ten reported cases, nine were males and one female. Eight of these patients died from direct effects of the local destruction of the disease. The bacteriologist's findings offer no help in the diagnosis. The ages of these patients were from 28 to 70 years.

Having seen recently a 40-year-old Mexican die with this same disease, I am convinced of the difficulty in making a diagnosis as well as the repulsive character of the lesion. He was probably seen by more physicians than any patient who had ever entered our hospital. Sections of the tissue were sent to pathologists throughout the United States and Canada, and as yet no definite diagnosis has been made. Regardless of our failure to diagnose or successfully treat such a lesion, it is well worth while to know that such a condition exists and that we are not alone in our failure.

Dr. Stewart is to be congratulated upon his clever and complete descriptive title, although it has no particular meaning in so far as the etiology of the lesion is concerned. The photographs and photomicrographs clearly illustrate the progress of the lesion as well as the inflammatory cellular reaction. Case reports are given in detail and an excellent bibliography is included. DIXON.

PHARYNX.

Bilateral Peritonsillar Abscess Treated by Tonsillectomy (Phlegmon bilateral de la loge amygdalienne traité par l'amygdalectomie totale "à tiède").

Canuyt, Lacroix and Wild (Strasbourg), Otorhinolar. Internat., 17:445 (June), 1933.

These authors systematically (1) made exploratory needle punctures followed by (2) wide opening into the tonsillar fossa, using local anesthesia. Then after two days to a week (3) the tonsils are removed. Three such double cases are reported, with no bleeding and rapid afebrile recovery. FENTON.

EAR.

Labyrinthine System and the Vasomotor and Respiratory Reflexes —Experimental Researches (Sistema labirintico e riflessi vasomotori e respiratori—Ricerche sperimentali)

Cantele, P. G. (Venice), Arch. Ital. di Otol., Rino. e Laryng., 44:129 (March), 1933.

The author publishes a very extensive article. He first reviews and discusses at length all the researches made, up to date, by the numerous workers, especially those of Spiegel and Demetriades and Camis; then he gives the purpose of his own investigations, the technic and the detailed descriptions of sixteen of the most characteristic experiments and fourteen graphs of the tracings, indicating methods and periods of stimulations, alterations in the blood pressure and respiration, also appearance and duration of nystagmus. Later he summarized all his experiments in nine distinct groups and compares the final results with those of Spiegel, Demetriades, Versteegh, Arndts and McNally, Foa, Luneder, Allers and Leidler and Camis.

The conclusions are as follows:

1. The caloric and mechanical irritation of the vestibular apparatus is followed by a reflex of negative blood pressure taken

either at the carotid or the femoral. Slight, inconstant and irregular variations in the respiratory movements were observed, which usually increased in rhythm with reduction in the amplitude of the excursions, especially during inspiration.

2. The fibers of the tympanic plexus passing over the promontory near the stapes, and posteriorly to the oval window, may participate in provoking the reaction of lowering the blood pressure. The route of transmission of this stimulus is supposed to be through the anastomotic fibers of the nervous intermedius.

3. The outflow of the perilymph from the vestibular cavity seems not to have any influence on the level of the blood pressure nor on the behavior of the respiration.

4. Mechanical destruction of the peripheral labyrinth is immediately followed by few and irregular oscillations in the respiratory curve, which slowly and persistently tend to diminish.

SCIARRETTA.

Report of Committee for the Consideration of Hearing Tests.

Journal of Laryng. and Otol., 48:22 (January), 1933.

The committee presented its report in three parts. In part 1 they considered the physical properties of tuning forks when they are used for testing hearing. Problems discussed in relation to the ear were its adaptation to an environment of accidental noise, auditory fatigue and the departures from the normal degrees of adaptation and fatigue which result from disease of the cochlea and the conducting mechanism of the ear. The original descriptions of the qualitative tuning fork tests were studied and recommendations were made as to how the tests are best carried out and the significance of the results. The committee discussed the advantages and disadvantages of Galton's whistle and the monochord for testing notes of higher pitch than are emitted by tuning forks. They recommend that the monochord be used.

Part 2 deals with further investigations of the effect of noise in testing and reports on quantitative tests. Recommendations are made in testing the hearing by speech. To eliminate the present discrepancies in recording the results of tuning fork tests the committee advises a standard method and adopts the "half intensity unit." The results of quantitative tests are expressed by the statement that hearing by air conduction or by bone conduction differs

by so many half intensity units from the normal. Forks with known half intensity periods are being manufactured with the period marked on the stem, and other forks can be calibrated as given in the report. The advantages and disadvantages of audiometers are discussed. The committee states that in their application to chemical otology, audiometers have failed, in that results obtained differ from those obtained from tuning forks. The results of testing by tuning forks have been for many years and still are the standard in otology.

Part 3 deals with audiography. The tabulation that results from the expression of quantitative tests is unwieldy and difficult of comparison, and the committee advises that a graphic method of expression should take its place. The time required to make a graph is not longer than that taken to record the qualitative tests and the hearing can be appreciated at a glance.

A scheme from the report is suggested as a guide to testing deafness. GOLDSMITH AND MCLEOD.

Otorrhea.

E. Watson Williams (Bristol), British Medical Journal, July 8, 1933.

Suppurative otitis media begins in early life and a large proportion during the first two years. Except as a complication of severe influenza, a first attack in adult life is uncommon. Two-thirds of the first attacks of otitis occur between the ages of 3 and 14 years. A chart and graph showing the age incidence of otitis media are given. It is believed from recent work that adenoids and teething catarrhs are symptoms of hypovitaminosis.

In the treatment of discharging ears all wet meatal applications are condemned. When the discharge is free the treatment advised is careful removal of this and application of powdered magnesium sulphate. In chronic cases it is indicated wherever the discharge is free or gelatinous, but it is in acute and subacute cases that its value is best seen. When the discharge is less persistent a powder of iodin 1 part and boric acid 100 is recommended. The author emphasizes the importance of early operation as a means of saving hearing. If, in a chronic case, the discharge continues offensive or frankly purulent despite six weeks of careful treatment, the cause is practically certain to be osteitis, as a rule with cholesteatoma. The question of operation should then be seriously

considered. In influenzal otitis, when there is considerable deafness, the question of operation assumes early importance, but if four weeks from the start of any acute otitis the ear has not healed, or at least not quite definitely healing, the decision as to operation must by no means be postponed. GOLDSMITH.

MISCELLANEOUS.

Some Disorders of Speech in Childhood—Their Nature and Treatment.

D. Guthrie (Edinburgh), Edinburgh Medical Journal, Vol. XL, No. 4, page 45.

A clinic for defects of speech has now been in operation for three years at the Royal Hospital for Sick Children, Edinburgh. The author deals with cases which require special training in breathing and in speaking after operations for cleft palate and adenoids and with stuttering, delayed speech and various other defects. The clinic is under the charge of a teacher who has been specially trained in voice production and in the treatment of disorders of speech.

The development of speech in normal children and the frequency of speech defects are reviewed. In the general population the proportion of stutterers is said to be 8 per 1000.

The three mechanisms of respiration, phonation and articulation in the production of normal speech are discussed.

A demonstration was given on the correct method of breathing for vocalization. The effect of physical or mental deficiency and deafness in relation to speech development are discussed. In the stutterer the mechanisms of respiration, phonation and articulation are all in good order, but there is a psychical cause and an attempt should be made to discover and correct it. The condition of overtension and fear of failure to speak correctly is found in all stutterers, and treatment must be directed against it, rather than against faulty articulation. The treatment of stuttering in children combines both psychologic and physical methods. They include a restful atmosphere in the class; complete muscular relaxation; helpful "suggestion"; breathing exercises and voice for rhythmic co-ordination; general encouragement and development of self-confidence and improvement of the child's environment.

GOLDSMITH.

The Treatment of Allergic Diseases in General Practice.

George W. Bray (London). The British Medical Journal, July 8, 1933.

Hypersensitiveness, or "allergy," on the part of an individual to many usually harmless articles commonly contained in his diet or environment manifests itself in such numerous and apparently diverse conditions as asthma, hay fever, eczema, urticaria and migraine, as well as in an ever-increasing host of minor affections.

It was at first thought that only protein substances could act as "allergens," but recent observations have shown that many non-protein factors, such as carbohydrates, drugs, and even physical agents, can lead to identical reactions. Allergic responses usually originate in one of two ways, either as a definite inheritance of the predisposition to manifest a specific hypersensitiveness of some type to some substance, or acquired as the result of tissue damage or excessive irritation. During childhood the chief etiologic factor is the hereditary tendency, but in the majority of adults postnasal factors have been particularly severe and constant; for example, the continual association with a particular allergen, either by occupation or environment, combined with, possibly, extensive tissue trauma. A division of cases, if possible, into primary (hereditary) and secondary (acquired) is of great importance in determining the causative allergen, as the latter group is practically always inhalant or occupational in nature. For this reason a thorough history of the time and mode of onset is essential.

The characteristics of allergic diseases and their treatment are discussed. A careful history will often indicate the cause, but in other cases no clue is given, and skin tests, or the use of elimination sheets are advisable. When skin tests are not practicable the patient should avoid the common agents that lead to attacks, that are either of an inhaled or ingested nature. The inhalants are the more common cause of allergic reactions at all ages except infancy, when foods predominate, and include feather, down, and horsehair pillows and bedding, animal pets, house dust and orris root.

When skin tests are not available, to determine causative foods, the simplest procedure is to adopt a diet which is free from the common causes of attacks. The author gives an example of a

basic diet on which a patient is placed until free from symptoms, then one of the excluded foods is added every few days until symptoms recur when generally the latest addition is the causative food. This does not mean that it is the sole food, for patients are generally multisensitive, so that the process must be continued until all the excluded foods are reintroduced except those that lead to reactions.

Contributory factors besides the removal of common specific causes are reviewed. Reflex irritation may precipitate asthmatic attacks, so that foci of infection should be removed. With regard to nasal abnormalities, it is essential to realize that they are commonly the result of allergic reactions, and rarely a primary cause. Whilst infected tissues should be removed or drained, boggy tissues will usually subside following therapy based on allergic principles. The causes and treatment of hay fever, infantile eczema, papular urticaria eneuresis and migraine are discussed. In many cases hay fever symptoms are present more or less all the year round and are due to causes other than pollens. These include the common inhalants, certain foods, drugs, of which aspirin is the commonest, and bacteria, though infection is the exception rather than the rule. Nasal cauterization and zinc ionization meet with occasional success, usually only temporary. Intranasal operations generally afford very little relief in the absence of sepsis. The curative treatment demands the active removal of sensitization factors, as determined by skin tests, or their passive avoidance by a change of climate or occupation. Desensitization is frequently necessary and when employed it should be specific—that is, against the causative allergens—if possible. GOLDSMITH.

Plaster Casts Showing the Relationship of the Structures of the Neck Following Strangulation, Homicidal or Suicidal.

Orton, Henry B. (Newark, N. J.). Trans. Am. Laryng. Assoc., 1933.

According to Dr. Harrison S. Martland of Newark, N. J., much confusion has existed concerning the mode of death in manual strangulation, throttling by other means and suicidal, of strangulation show that the main factor is asphyxiation due to the shutting off of the air passages, and that all other factors are practically always of secondary or contributory importance.

To prove this contention plaster models showing the anatomic relationship of the structures in the mouth, nasopharynx and accidental and homicidal hanging. Observations in these types glottis, were presented. IMPERATORI.

Causation and Treatment of Arthritis and Allied Conditions.

Report of the B. M. A. Committee, The British Medical Journal, June 17, 1933.

The Council of the British Medical Association appointed a special committee for the promotion of research into the causes of arthritis and allied conditions and the investigation of methods of treatment.

The report is given under fourteen different headings and covers nineteen pages.

1. Nomenclature and Classification.—The committee adopted a clinical grouping of cases for its investigation, as an etiologic classification is in the present state of knowledge impossible. It excluded from the scope of inquiry the joint affections of acute and subacute rheumatism; specific infections such as those dependent upon gonococcal and pyogenic organisms; metabolic and hemopoietic diseases and organic nervous diseases.

2. Clinical Features.—The symptoms, prognosis and treatment of each clinical group are reviewed.

(a) Primary rheumatoid arthritis which occurs particularly during the child-bearing period in women who follow indoor occupations, commences in the small joints of the hands and develops a characteristic fusiform shape without much evidence of active inflammation. Prognosis is always slow, and intermissions and recrudences are frequent.

(b) Secondary rheumatoid arthritis is divided into two clinical styles: (1) with acute febrile onset resembling rheumatic fever; (2) with subacute onset, often followed by periods of activity and remission. In each style the disease is polyarticular, tends to be symmetrical in distribution, and attacks both large and small joints. The disease is usually associated with discoverable septic foci. Many patients, especially if treated in the early stages, recover completely after removal of septic foci, whereas in others this procedure is of no avail. In some instances general treatment

is followed by improvement both of the arthritis and of the infective focus without removal of the latter.

(c) Chronic Villous Arthritis.—The early symptoms are gradually increasing stiffness and pain in the affected joints (usually the knees), often at the onset, worse during rest. Later walking, especially on rough ground, becomes difficult. With adequate local and general treatment the prospect is favorable.

(d) Osteo-arthritis is distinguished by primary degenerative changes in the articular cartilages, followed by bony overgrowth in the neighborhood of the joints, namely, the large joints.

(e) Spondylitis.—(1) Spondylitis ankylopoietica is characterized by a tendency to ankylosis of the vertebral and adjoining articulations; (2) spondylitis osteo-arthritis is similar to osteo-arthritis in other parts of the body, with degenerative changes in the bones and compensatory osteophytic outgrowths.

(f) Fibrositis is an inflammation of the connective tissues of the body. It may effect the muscle sheaths, the deep fasciæ or the fibrous portions of the joint capsules. The main varieties are (a) Intramuscular, fascial and peri-articular fibrositis and bursitis; (b) panniculitis—that is, fibrositis of the subcutaneous tissue; (c) perineuritis.

3. Incidence.—No accurate statistical record of the diseases discussed is available, but the National Health Insurance Service estimates that the loss of work time in a single year for arthritic diseases alone is 4,500,000 days and the cost involved under the National Health Insurance Scheme is 4,500,000 pounds sterling.

4. Etiology.—In primary rheumatoid arthritis the etiology is uncertain, while in the secondary form infection plays an important part. In chronic villous arthritis evidence of infection is inconclusive; thyroid deficiency with strain on the affected joint due to excessive bodily weight is important. In osteo-arthritis infection makes some contribution in secondary cases, but in the primary form strain and arterial degeneration (local and general) are often provocative agencies. In spondylitis ankylopoietica infection is the predominant cause. In fibrositis many cases are due to infection, but perhaps with equal frequency the cause is metabolic disorder and defective elimination.

5. Morbid Anatomy.—A description is given of the pathologic processes in each clinical group of cases under discussion.

6. Biochemistry.—The sedimentation rate depends on the rate at which red blood corpuscles sink under the influence of gravity when oxalated blood is drawn off into glass tubes. In all infective conditions there is a tendency for sedimentation to be more rapid than the normal rate. The findings in the different groups of arthritis are given. Other blood tests as well as analysis of the urine and gastric juice are discussed.

7. Radiology.—The radiographic changes in each group of arthritis are reviewed. It is believed that radiology may be expected to supply an increasingly useful and direct means of differential diagnosis in case of chronic arthritis, especially as the more accurate methods now being employed enable the minute changes which characterize the early stages of the disease to be recognized and recorded.

8. Differential Diagnosis.—The differentiation of the forms of arthritis dealt with in the report as well as separating them from other varieties of arthritis is given. To distinguish the pain and limitation of movement due to arthritis or fibrositis from similar symptoms produced by other diseases is also considered.

9. Treatment Prophylaxis.—Multiple factors that may be concerned in the etiology of these diseases as well as conditions which impair the general health and induce metabolic disorders must be taken into account.

As the precise etiology of none of the forms of arthritis under review has yet been determined with certainty, vaccine therapy does not rest on so secure a foundation as it does in conditions where the causative agent is beyond dispute. Taken as a whole, however, the evidence now available with regard to the bacteriology of arthritis indicates that organisms of the streptococcal group are in some way concerned in its etiology, and further evidence in favor of this view is furnished by the beneficial results of vaccine therapy in certain cases.

Drugs have no specific value but they are of service in the relief of pain and in improving the patient's resistance and general well being. Endocrine disturbances are in general not sufficient by themselves to produce joint diseases, but in a considerable propor-

tion of cases they create a suitable soil for other factors to produce effects harmful to the joints. In confirmation of this is the general experience that endocrine therapy may often bring about improvement, but will rarely completely cure a joint disease. Attention may be confined to the thyroid and ovaries, as any influence that the other endocrine glands may have is very problematical.

There is a great diversity of opinion regarding diet. Some patients appear to suffer more pain or stiffness after articles of food termed "acid," while others do well on these substances. The diet should have its due proportion of each of the essential vitamins. In fibrositis and chronic villous arthritis excess of the carbohydrates must be avoided. Physical treatment such as the application of heat, massage and electrical methods as well as mineral waters and baths are discussed.

10. Orthopedic and Surgical.—The deformities which develop and the correct care and position of the different joints during the disease are reviewed. Surgical measures, whether by fixation, manipulation under an anesthetic or operation, are discussed.

11. National schemes for treatment in Holland, Germany and Sweden are reviewed and a suggested national scheme for Great Britain is given.

12. Observation on Advertised Remedies.—Attention is called to proprietary remedies and unqualified treatment for the various conditions of arthritis. The need for legislation for the control of the traffic in proprietary remedies and provision to enforce such control is emphasized.

13. Indications for Future Research.—The incidence of arthritic conditions in different trades and occupations and in various localities under different climatic conditions should be determined. Indications for research work in the bacteriology and biochemistry are reviewed. Controlled experiments are needed to determine the value of the numerous therapeutic methods advised for the treatment of the various forms of arthritis.

14. Suggestions for Further Professional Education.—The committee recommended that further postgraduate work and lecture demonstrations on the diagnosis and treatment of arthritis should be given. GOLDSMITH.

Radium Burns.

J. Mervyn Thomas (Cardiff), The British Medical Journal, July 8, 1933.

The author discusses radium burns during or following surface radiotherapy by the plaque method. The skin undergoes certain changes during irradiation, beginning with an erythemia towards the third day, and passing through an edematous stage to tissue necrosis somewhere about the sixth to tenth day. The edematous stage should be the aim of the therapist during treatment and if it is then stopped the skin rarely ulcerates.

Radium treatment should not be given to patients suffering from cachexia, anemia, a white cell count under 5,000 per cm., diabetes, nephritis, extreme obesity, advanced myocarditis, active pulmonary tuberculosis, marked arteriosclerosis, or any general debilitating disease, general sepsis, local inflammation or infection. There must be no ulceration of the skin immediately prior to treatment. The skin must be cleansed and lightly powdered with dusting powder before applying the plaque, and this must be repeated on alternate days to ensure the absence of perspiration or fetid discharge irritating the skin. The plaque should fit the area to which it is applied, firmly and without possibility of friction, and removed at once on the appearance of edema in the skin. The part should be frequently well powdered or calamine lotion applied. No soap should be used for two months. When a burn appears the area should be cleansed thoroughly without soap and a dressing of eucalyptus and paraffin (5 per cent) applied. This should be replaced by sterile vaselin at a later stage when the healing is evident. The use of powder should be avoided at this stage. When healing is intractable, rapidity may be aided by an erythema dose of ultra-violet light. Three illustrative cases are given in this article. GOLDSMITH.

Books Received.

Problems of the Deaf.

Max A. Goldstein, M. D., F. A. C. S. Leather. 610 pages with 273 illustrations. St. Louis: The Laryngoscope Press: 1933. Price $6.00.

This is the most comprehensive and best contribution to the problems of the deaf that has been published. Every otolaryngologic library should contain this volume. The chapter on "Hearing Devices" is well worth the cost of the book to the otolaryngologist.

Every general physician should read the chapters dealing with "The Deaf Child," the "Hard-of-Hearing Child," "The Physician and the Deaf Child," and the "Parent of the Deaf Child."

This book should be available to all teachers in the public schools. The chapter on "The Hard-of-Hearing Child" is particularly valuable to teachers. The chapter dealing with "Schools for the Deaf" will render the greatest assistance to persons interested in helping the deaf child to get an education..

La Reeducation des Obstrués du Nez et du Rhino-pharynx (Reeducation of Mouth Breathers).

Geo. Rosenthal, M. D. (Paris). Paper. Sm. 8vo of 128 pages with 36 illustrations. Paris: G. Doin: 1933. Price 28 francs.

An elementary manual rewritten from lectures given thirty years ago, primarily for general practitioners, teachers and those charged with the care of institutionalized children; brought thoroughly up to date and including modern views on forced breathing; but lacking any consideration of orthodontic or surgical relief. FENTON.

Courts and Doctors.

Lloyd Paul Stryker. Cloth. 12mo of 236 pages. New York: The MacMillan Company, 1932. Price $2.00.

From a background of many years' experience as chief counsel for the New York State Medical Society, the author has produced a valuable book for the busy doctor or student of medicine.

The seven parts are devoted to major subjects, such as: The Practice of Medicine, Relation of Physician to Patient, Expert

Testimony, the Doctor and Criminal Law, and Actions for Malpractice. In the thirty chapters, the numerous medicolegal pitfalls and problems are clearly discussed and illustrated by brief case reports and court decisions.

So well written is the entire book that it reads more as a narrative, and is well worth its few hours' reading time. VOTAW.

L'Oreille et ses Maladies; l'Audition et ses Troubles; les Organes de l'Équilibre (Diseases of the Ear; Disturbances of Hearing; the Vestibular Organs).

A. Bonain, M. D. (Brest). Paper. Large 8vo of 294 pages with 194 illustrations (20 colored). Paris: G. Doin: 1933. Price 45 francs.

This brief treatise includes not only much of the historical background of otology, but also the incisive and brilliant personal opinions of one of the most original French clinicians, set off by remarkably clear and accurate line engravings.

He relies on Shambaugh's pioneer work for much of the minute structure of the internal ear. An excellent résumé of present views on aural physiology contains a comparative table of the theories of ten investigators. Bonain holds that "sorting out" of tones occurs peripherally, their interpretation centrally. By omitting details of surgical technic, he is able to discuss diagnosis and operative indications most completely. Master of local anesthesia, his formulæ for the management of external otitis and intratympanic conditions are classical.

This book contains in short compass much matter of great value to teachers of otology, and many useful therapeutic hints.

FENTON.

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